THE

BRITISH JOURNAL

OF

CHILDREN'S DISEASES.

VOL. XI. SEPTEMBER, 1914. No. 129.

Original Articles.

CARDIAC ARRHYTHMIA IN DIPHTHERIA.

By E. B. GUNSON, M.D.Edin., M.R.C.P.Lond.,

Research Assistant, Cardiac Department, London Hospital; Late Assistant Medical Officer, Grove Fever Hospital, London.

INTRODUCTION.

IN health the ventricles respond regularly to rhythmical impulses which arise in the sino-auricular node and pass through the auricle to the auriculo-ventricular node and along the bundle of His and its branches.

Sinus arrhythmia.—Normally there occurs a slight quickening of the pulse during inspiration and a slowing during expiration, producing a definite irregularity known as sinus arrhythmia. This variation, due to vagal influence, is induced by respiratory movement and is constant in children and young adults, but generally absent in later life. Sinus arrhythmia, when extreme, may give rise to marked irregularity of the pulse, but it is readily recognised owing to its constant relationship to respiration (see Fig. 1).

Pathological irregularities of the pulse.—These may be divided into two groups: (1) Irregularities dependent upon abnormal impulse formation. These include the majority of irregularities, and may be due to premature contractions, auricular flutter or auricular fibrillation.

(2) Irregularities dependent upon interference in conduction, or heart block.

(1) Abnormal impulses may arise from the heart muscle itself or from the tissue at the junction of auricles and ventricles, the auriculo-ventricular node. The contractions of the ventricles to

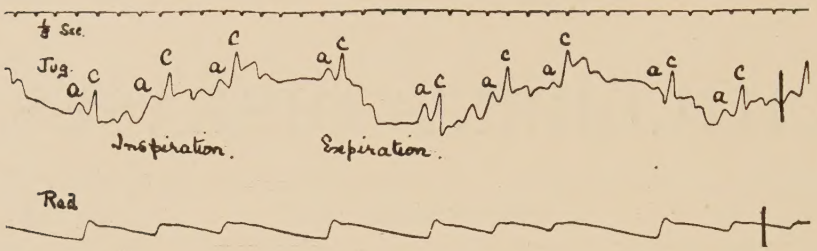

FIG. 1.—Marked sinus arrhythmia. Showing the relationship of the arrhythmia to respiration; the pulse quickens during inspiration and slows during expiration. Rate 75 per minute.

which they give rise are premature and are known as extra-systoles; auricular, ventricular or atrio-ventricular extra-systoles according to their origin. Extra-systoles or premature contractions produce premature beats at the wrist, with consequent irregularity of the pulse. Premature contractions are recognised on auscultation by the fact that the heart sounds to which they give rise immediately succeed

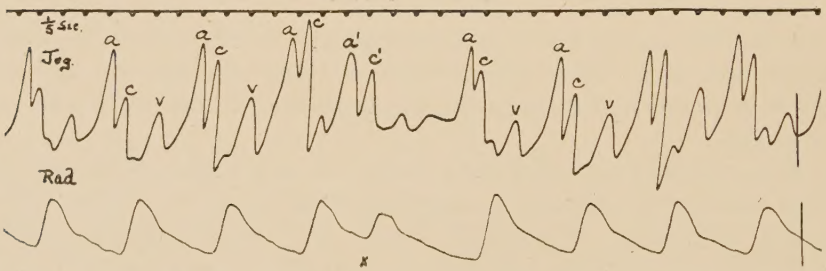

FIG. 2.—A single premature auricular contraction. The premature contraction at X is followed by a pause longer than that occurring between two normal beats. In the jugular curve the *a–c* interval for the premature contraction is greater than $\frac{1}{5}$ sec. Rate 79 per minute.

those of the preceding contraction and are followed by a pause greater than that occurring between two normal beats. As a rule they have no definite relationship to respiration (see Fig. 2). The varieties of premature contractions can only be differentiated by means of graphic records. Premature contractions commonly occur

singly, occasionally in short runs. Premature contractions may, however, initiate a new and more rapid rhythm composed of a succession of such premature contractions, with the production of an auricular, ventricular or atrio-ventricular tachycardia as the case may be. Following Lewis, the term "atrio-ventricular" is substituted

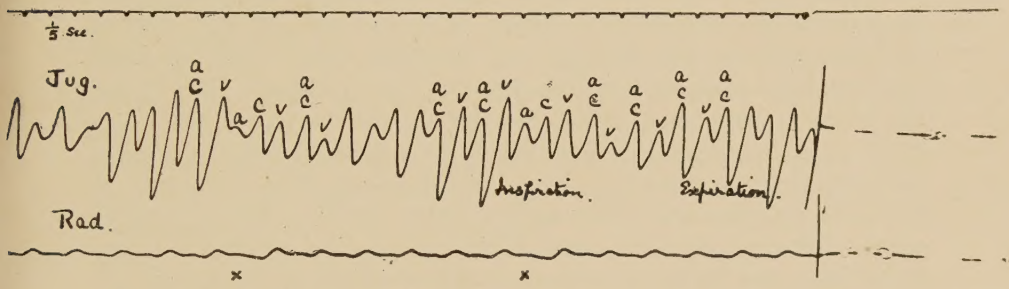

FIG. 3.—CASE 1.—Thirteenth day, 6 p.m. Atrio-ventricular rhythm, showing a return, under vagal influence, to the normal rhythm at x, maintained for one beat only in each respiratory cycle. Rate 160 per minute.

throughout the present paper for the word "nodal" on the ground that the latter is not sufficiently distinctive.

A special variety of auricular tachycardia has been described under the term auricular flutter.

Auricular fibrillation is a condition in which the normal auricular

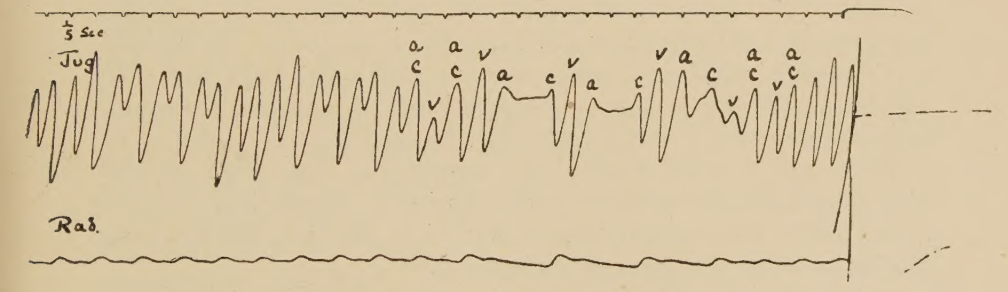

FIG. 4.—CASE 1.—Thirteenth day, 6.30 p.m. Offset and onset of atrio-ventricular rhythm, with a rate of 180 per minute. It shows three intervening beats at approximately half the rate. In these the *a–c* interval varies from $\frac{2}{5}$ to $\frac{1\cdot5}{5}$ sec. producing three radial beats of different length.

contractions are replaced by rapid fibrillary movements of the auricle. The ventricular response is haphazard and gives rise, except on very rare occasions, to complete irregularity of the pulse.

Pulsus alternans is the term applied to an irregularity of the pulse in which each alternate beat is smaller than the one preceding.

The pause leading up to the small beat is either equal to or greater than that which follows the same beat, thus differentiating the condition from the irregularity due to the presence of premature contractions.

(2) Supra-ventricular impulses may either be delayed or entirely blocked on their way to the ventricle. Heart block is not a common cause of pulse irregularity. Delay in conduction, regular degrees of block such as 3:1 and 2:1 and complete heart block all occur with complete regularity of the pulse. Only in those cases where the block is infrequent and in the nature of "dropped beats" or where such degrees of block as 3:2 or a "mixed" block are present, does the pulse become irregular. Sudden and great reduction in the pulse rate is usually the result of heart block. For a detailed account of these conditions and of the pathological processes underlying them, the reader is referred to the writings of Mackenzie (5) and of Lewis (4).

Historical.

In 1910, Magnus-Alsleben (6) published tracings showing the presence of complete heart block, which developed on the fourth day of disease in a case of severe faucial and laryngeal diphtheria. The heart block persisted until death on the ninth day.

Fleming and Kennedy (1) in the same year reported a similar case.

Rohmer (8), in 1911, published electrocardiograms of two fatal cases showing complete heart block. The same author (9), in 1912, reported five additional fatal cases in which the pulse remained regular. Two cases showed complete heart block, three cases were possibly examples of atrio-ventricular rhythm.

Price and Ivy Mackenzie (7) described a fatal case of complete heart block, associated with auricular fibrillation. In the absence of more conclusive proof than that afforded, the auricular fibrillation must be regarded as doubtful.

Sperk and Hecht (10), in 1912, reported a case of 2:1 heart block.

Hecht (2), in a recent monograph on the heart in childhood, deals at some length with cardiac disorders in diphtheria. He obtained electrocardiograms from forty-two cases, while at least an equal number, which clinically gave no evidence of cardiac involvement, were not electrocardiographed. His series included one case of 2:1 heart block, appearing during the first week and persisting four months later and one case of atrio-ventricular rhythm which

persisted for several days, when there was a return to normal rhythm interrupted by extra-systoles, auricular, ventricular, and atrio-ventricular. Recovery took place. A third case presented, on the sixteenth day of disease, frequent premature contractions which persisted until death on the thirty-fifth day from collapse of lung secondary to diaphragmatic palsy.

Hume (3) reported three fatal cases and one which recovered. Of the fatal cases two showed premature auricular contractions on the ninth day, atrio-ventricular or nodal rhythm the following day and a return to normal rhythm, interrupted by premature auricular contractions, before death. An intervening rhythm of uncertain nature was regarded tentatively as auricular flutter. In a third case, also presenting premature auricular contractions and atrio-ventricular rhythm, 2:1 heart block developed three days before death. In the fourth case, in addition to premature auricular contractions, paroxysms of auricular tachycardia occurred; the patient ultimately recovered.

Thus, in eighteen recorded cases of diphtheria in which graphic records of the heart's action were obtained, the following conditions were present: Auricular premature contractions, five cases; auricular tachycardia, one case; ventricular premature contractions, one case; atrio-ventricular premature contractions, one case; atrio-ventricular rhythm, seven cases; heart block, 2:1, three cases; heart block, complete, six cases.

In practically all the fatal cases the myocardium was the seat of fatty change; in two, definite myocarditis was present. In only two of the six cases of complete heart block investigated microscopically was there a lesion of the auriculo-ventricular node or bundle. In three cases of atrio-ventricular rhythm the auriculo-ventricular node and bundle were healthy; the sino-auricular node was the seat of an inflammatory process in two of the cases.

The frequent failure to discover changes post-mortem to account for the conditions during life is noteworthy. Many exhaustive investigations into the condition of the heart muscle and conducting tissues in fatal cases of diphtheria, notably those of Tanaka (11) have produced similar negative results.

Present Observations.

The present paper is based on more than 750 polygraphic curves obtained from children under ten years of age, in whom the diagnosis of diphtheria was confirmed bacteriologically: 120 cases

were investigated. Polygraphic tracings were taken in all the severe cases, with a single exception, at least once a day from the time of admission. Twenty-five consecutive mild and moderately severe cases were traced on alternate days throughout their stay in hospital, a period varying from five to eight weeks, in order to

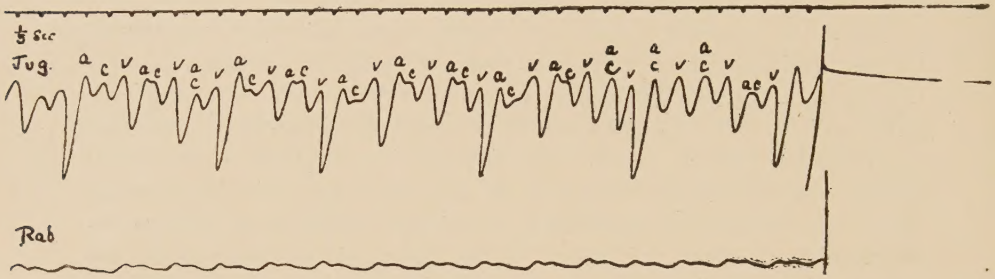

FIG. 5.—CASE 1.—Fourteenth day. Arrhythmia due to a varying *a–c* interval, which shows transition stages from almost $\frac{1}{5}$ sec. to true atrio-ventricular beats where *a* and *c* fall together. The variation in the length of the radial beats corresponds to the variation in the length of the *a–c* intervals. Rate 133 per minute.

determine the incidence and the nature of the arrhythmia in this type of case.

Antitoxin.—Mild cases received on admission 8000 units, cases of moderate severity 12,000 to 16,000 units, severe cases 16,000 to 20,000 units. In moderate and severe cases the antitoxin was

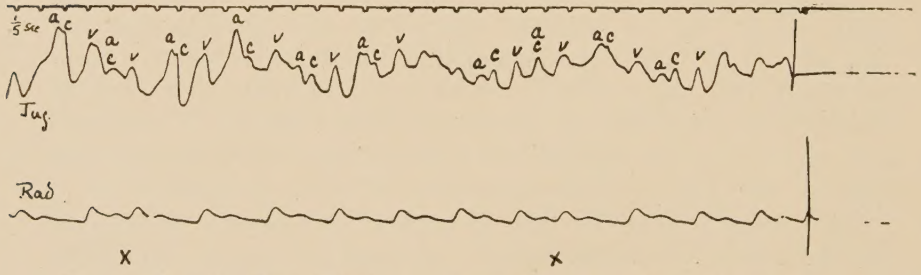

FIG. 6.—CASE 2.—Twentieth day. Premature atrio-ventricular beats at x, followed in each case by a second atrio-ventricular beat with an *a–c* interval less than $\frac{1}{10}$ sec. Rate 116 per min.

repeated once or twice. Intra-muscular injections were given in each case.

Method of investigation.—The records were taken with a Mackenzie polygraph. By this means simultaneous tracings of the jugular and radial pulses were obtained, with a time-marking record. The observations were made about the same time each afternoon.

Method of analysing tracings.—The jugular curve is marked by comparison with the radial. The distance from the index mark to any given radial upstroke is measured by dividers. To this is added a space representing $\frac{1}{10}$ sec., this being the transmission time from the neck to the wrist. One limb of the dividers is now placed

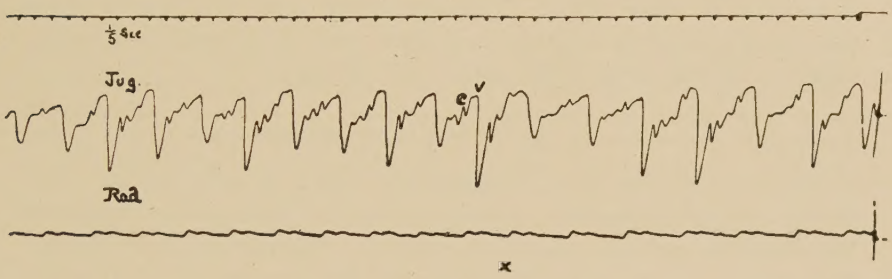

FIG. 7.—CASE 3.—Twenty-fifth day. Showing a change in rate at x. The nature of the rhythm is doubtful as the *a* waves cannot be accurately determined. Rates 105 and 83 per minute.

against the index mark of the jugular curve, the other limb when brought to rest on the jugular curve indicates the upstroke of the rise due to the corresponding carotid pulsation, *c*.

The rise *v* is due to venous stasis before the auriculo-ventricular valves open. The fall of this wave corresponds exactly to the

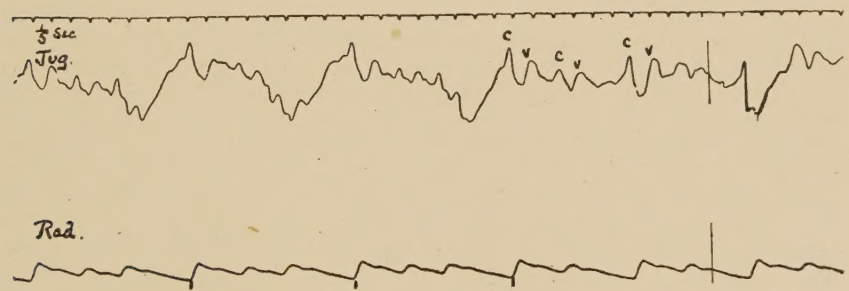

FIG. 8.—CASE 3.—Thirty-seventh day, 7.5 p.m. Frequent premature auricular contractions occurring singly and in pairs. Rate 105 per minute.

dicrotic notch on the radial curve. The rise occurring immediately before *c* is due to auricular systole and is called the *a* wave. The space between these two waves, the *a–c* interval, represents the time occupied by the passage of the impulse from the auricle to the ventricle. Normally the *a–c* interval measures $\frac{1}{5}$ sec. or less.

A.—Type of Arrhythmia in Mild and Moderately Severe Cases.

Apart from normal sinus arrhythmia, the only type of irregularity met with in these cases was due to premature auricular contractions, which occurred in 28 per cent. of cases. They appeared as early as the ninth and as late as the sixty-sixth day of disease; their average duration was three days. They produced only slight arrhythmia and were ignored in treatment, as they were unaccompanied by any other symptoms or signs of cardiac involvement.

B.—Type of Arrhythmia in Severe Cases.

Three cases, fatal from toxæmia, developed no arrhythmia. Five cases developed so-called "cardiac paralysis," with gallop rhythm on auscultation, cardiac dilatation, hepatic enlargement and marked arrhythmia. This arrhythmia was due to the presence of frequent premature contractions, and in two cases these were followed by paroxysms of tachycardia. The nature of the arrhythmia is discussed in each case, together with the other physical signs present at the time.

The term gallop rhythm as applied to the triple character of the heart sounds on auscultation has been used in the descriptions of the severe cases. The fact that such auscultatory phenomena may be present with a regular pulse renders the term undesirable.

Case 1.—"*Cardiac Paralysis.*" *Premature atrio-ventricular contractions. Atrio-ventricular rhythm. Prolonged a–c interval. Death.*

D. M—, female, aged 7 years, was admitted on the third day of disease suffering from severe faucial diphtheria with considerable œdema of the fauces and subcutaneous œdema of the neck. The pulse remained regular for ten days with a rate varying from 130 to 84 per minute. Sinus arrhythmia was almost constant. On the tenth day gallop rhythm was present on auscultation and persisted until death. The pulse was regular but feeble. On the thirteenth day the pulse became irregular. At 4 p.m. the rate was 122 per minute; the irregularity was due to the presence of beats of varying length with a corresponding variation in the length of the *a–c* interval. The long beats had an *a–c* interval of more than $\frac{1}{5}$ sec., that of the shorter beats varied from $\frac{1}{5}$–$\frac{1}{10}$ sec. The variation had no constant relationship to respiration. Deep cardiac dulness extended 1 in. to the right of the middle line and 1 in. beyond the left nipple line. Liver dulness extended $1\frac{1}{2}$ in. below the costal margin. At 6 p.m. the pulse-rate had increased to 160 per minute and atrio-ventricular rhythm was present, interrupted at the beginning of each respiratory cycle by a long beat, corresponding to which in the jugular curve was a normal *a–c–v* sequence. This suggested the return, under vagal influence, to a normal rhythm, maintained for one beat only in each respiratory cycle (see Fig. 3). At 6.5 p.m. atropin, $\frac{1}{200}$ gr., was injected subcutaneously. During the next hour there was frequent interchange between the irregular rhythm of 4 p.m. and an atrio-ventricular tachycardia at the rate of 180 per minute. The former predominated for the first fifteen minutes, the latter subsequently (see Fig. 4). At 11.30 p.m. the pulse was feeble and very irregular, with a rate of 100 per minute. The *a–c* interval was variable and occasional

atrio-ventricular premature contractions occurred. On the 14th day the rhythm was chiefly atrio-ventricular. At 4 p.m., two hours before death, the pulse was irregular, with a rate of 133 per minute. The *a–c* interval showed various transition stages from almost $\frac{1}{5}$ sec. to true atrio-ventricular beats where *a* and *c* fell together, the variation in the length of the radial beats corresponding exactly to the variation in the length of the *a–c* interval (see Fig. 5).

CASE 2.—"*Cardiac Paralysis.*" *Premature atrio-ventricular contractions. Recovery.*

V. B., female, aged 6 years, a case of severe hæmorrhagic diphtheria, was admitted on the 5th day of disease. On the 15th day the pulse became irregular from the presence of frequent premature atrio-ventricular contractions. Deep cardiac dulness extended 1 in. to the right of the sternum and 1 in. outside the left nipple line. Gallop rhythm was present on auscultation and persisted until the 20th day, the arrhythmia, however, remained until the 22nd day, with no change in cardiac dulness. The pulse at this time was feeble and the rate 116 per minute (see Fig. 6). On the 23rd day the pulse again became regular and remained so throughout convalescence.

CASE 3.—"*Cardiac Paralysis.*" *Frequent premature auricular contractions. Auricular tachycardia. Recovery.*

E. C., female, aged 6 years, a case of severe faucial diphtheria, was admitted on 3rd day of disease. From the 11th to 16th days the pulse was regular, the rate varying from 72 to 52 per minute. On the 17th day the pulse became irregular from the presence of occasional premature auricular contractions. Cardiac dulness extended 1 in. to the right and 4 in. to the left of the mid-line. Gallop rhythm was present on auscultation. Liver dulness extended $1\frac{1}{2}$ in. below the costal margin. There was some restlessness and occasional cyanosis. This irregularity of the pulse persisted for eight days. On the 25th day frequent paroxysms of increased rate occurred. For two days two distinct rhythms alternated. The one with a pulse rate of 80 to 75 per minute was occasionally interrupted by premature contractions. The other was a regular rhythm and varied in rate from 125 to 105 per minute (see Fig. 7). Cardiac dulness was still increased and gallop rhythm persisted on auscultation. The liver extended $3\frac{1}{2}$ in. below the costal margin, was painful and very tender. There was extreme restlessness. On the 27th day the more rapid rate, 135 to 120, became constant but was interrupted by premature contractions. Cardiac and liver dulnesses were reduced and the patient was easier. On the 37th day, pneumonia having supervened, the patient again became restless. Deep cardiac dulness extended 1 in. to the right and $4\frac{1}{2}$ in. to the left of mid-line. The liver extended 2 in. below the costal margin. At 7 p.m. the rhythm was normal, the pulse was regular with a rate of 88 per minute. At 7.5 p.m. the pulse became irregular from the presence of premature auricular contractions (see Fig. 8). The premature contractions became more frequent and led up to a paroxysm of auricular tachycardia, with a rate of 140 per minute, lasting for 30 minutes. Slight pulsus alternans was present (see Fig. 9). The following day the pulse was regular, with a rate of 110 per minute, and an *a–c* interval of $\frac{1}{5}$ sec. No further irregularity of the pulse occurred and the patient was discharged on the 100th day.

CASE 4.—"*Cardiac Paralysis.*" *Frequent premature contractions. Death.*

A. H., male, aged $8\frac{1}{2}$ years, was admitted on the 5th day of disease with very extensive false membrane, considerable faucial œdema and subcutaneous œdema of the neck. The pulse was feeble but remained regular for two days. The rate was 70 per minute, the *a–c* interval was less than $\frac{1}{5}$ sec. and slight sinus arrhythmia was present. On the 8th day the pulse became irregular from the presence of frequent premature contractions, occurring singly and in pairs. The pulse rate was unchanged (see Fig. 10). Cardiac dulness extended 1 in. beyond the left nipple line; there was

no increase to the right of the sternum. First and second sounds were present in all areas. The patient was very restless and extremely pale. On the 9th day at noon the radial pulse was almost imperceptible. There was marked pulsation in the neck

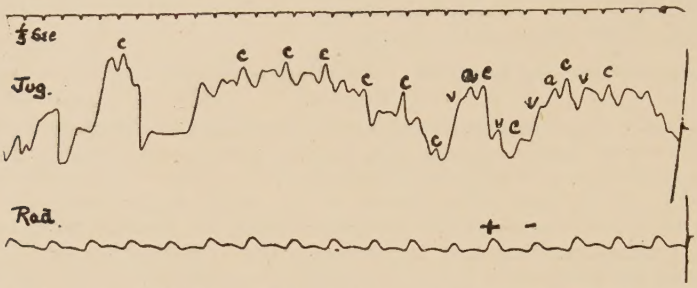

FIG. 9.—CASE 3.—Thirty-seventh day, 7.15 p.m. Auricular tachycardia showing slight pulsus alternans. Rate 140 per minute.

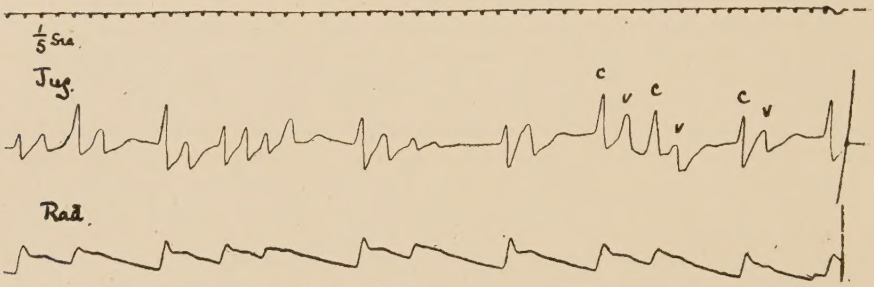

FIG. 10.—CASE 4.—Eighth day. Frequent premature contractions occurring singly and in pairs. The nature of the premature contractions is doubtful. There is no *a* wave in the jugular curve and the pauses following the premature contractions are either equal to or less than those following normal beats.

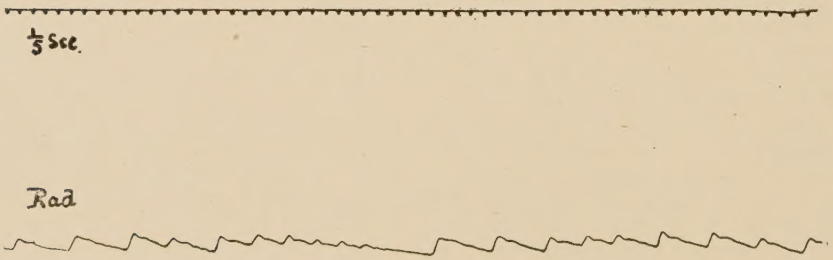

FIG. 11.—CASE 5.—Sixth day. Runs of premature contractions. Here also the nature of the premature contractions is uncertain.

and a jugular tracing taken one hour before death showed the persistence of the same irregularity. Gallop rhythm was present on auscultation and the liver extended 2½ in. below the costal margin. The nature of the premature contractions in this case is uncertain. It is difficult to explain the absence of *a* waves when the *c* and

v waves are so prominent and in a case where venous pulsation in the neck was well marked.

CASE 5.—"*Cardiac paralysis.*" *Frequent premature contractions. Death.*

D. F—, female, aged 8 years, was admitted on the fifth day of disease in a similar state to the preceding patient (A. H—). The pulse was regular, with a rate of 102 per minute. The *a–c* interval was normal. On the sixth day the patient was in a low semi-conscious state and there was suppression of urine. At 2.20 p.m. the pulse became irregular from the presence of premature contractions, the irregularity persisting for three hours (see Fig. 11). At 5.30 p.m. the pulse again became regular and remained so until 8 p.m., when it became imperceptible at the wrist, death occurring an hour later. Gallop rhythm on auscultation developed with the arrhythmia, but there was no increase in cardiac dulness. The nature of the premature contractions in this case also is doubtful. The probability is they were auricular in origin.

CASE 6.—*Toxæmia. No arrhythmia. Death.*

H. H—, male, aged 7 years, was admitted in a toxic state on the third day of disease and died the following day. On admission no jugular tracing was obtained owing to his extreme restlessness. The radial pulse was regular with a rate of 130 per minute. Six hours before death the pulse was regular and the rate 62 per minute. Slight sinus arrhythmia was present. No *a* waves were present in the jugular curve. There was no increase in cardiac dulness, and first and second heart sounds were present throughout. Liver dulness extended 1 in. below the costal margin.

CASE 7.—*Toxæmia. No arrhythmia. Death.*

A. H—, female, aged 4½ years, a similar case to the preceding, was admitted on the fourth day of disease and died on the 6th day. The pulse remained regular, with a rate which varied from 144 to 90 per minute. The *a–c* interval was less than $\frac{1}{5}$ sec. Cardiac dulness extended 1 in. to the right of the mid-line and first and second sounds were present in all areas. Liver dulness extended 1 in. below the costal margin.

CASE 8.—*Toxæmia. No arrhythmia. Death.*

S. W—, male, aged 8 years, a similar case to the two preceding ones, was admitted on the third day of disease and died the same night. Four hours before death the pulse was imperceptible at the wrist and a jugular tracing could not be obtained owing to the restlessness of the patient. The heart was regular. The rate at the apex was 190 per minute and the heart sounds were tic-tac in character. The apex beat was in the 5th space 1 in. outside the left nipple line. There was no increase in cardiac dulness to the right.

PATHOGENY.

The pathological changes underlying the condition known as "cardiac paralysis" are obscure. The associated arrhythmia would appear to depend upon abnormal impulse formation. In one case in the present series there was evidence that the centre of impulse formation was unstable and varied in situation between the sino-auricular node and the auriculo-ventricular node, the latter ultimately becoming the dominant centre for a new rhythm. The variation in the sequence of auricular and ventricular systoles in this case was so rapid that it suggests the presence of a disordered nervous control.

The Practical Significance of Cardiac Arrhythmia.

It is highly important that the varieties of cardiac arrhythmia met with in diphtheria should be recognised. Sinus arrhythmia, especially during the period of slow pulse rate occurring after the second week, may produce very marked arrhythmia and give rise to needless anxiety (see Fig. 1). Premature auricular contractions occurring singly and infrequently produce an arrhythmia which is unaccompanied by signs of cardiac inefficiency (see Fig. 2). Patients exhibiting either of these irregularities may, so far as the cardiac condition is concerned, safely be allowed up at the end of the fourth week and should not be detained in bed on account of the arrhythmia. On the other hand, the arrhythmia associated with the condition known as "cardiac paralysis," a syndrome including cardiac dilatation, gallop rhythm on auscultation and hepatic enlargement, and accompanied in most cases by restlessness, is of very grave significance, three of the five cases presenting this arrhythmia, in the present series, proving fatal. This arrhythmia is due to frequent premature contractions and is accompanied in some cases by paroxysms of tachycardia. The condition is an indication for absolute rest. Drug treatment is of doubtful value.

Summary.

(1) The irregularities of the pulse occurring in 120 cases of diphtheria in children under 10 years of age were investigated by means of the polygraph.

(2) Sinus arrhythmia was constant with pulse rates below 100 per minute.

(3) Premature auricular contractions, occurring singly and infrequently, were present in 28 per cent. of cases investigated systematically and constituted the sole irregularity in mild and moderately severe cases.

(4) Three cases, fatal from toxæmia, developed no arrhythmia and no heart block.

(5) Five cases, of which three were fatal, developed so called "cardiac paralysis" with marked arrhythmia due to very frequent premature contractions. In two of the cases the premature contractions led up to paroxysms of tachycardia, and in one case the tachycardia was associated with an arrhythmia dependent upon a changing a–c interval, which varied from $\frac{2}{5}$ sec. to less than $\frac{1}{10}$ sec.

(6) Premature ventricular contractions, auricular flutter, auricular fibrillation and heart block—apart from a prolonged *a–c* interval—did not occur in this series.

(7) No case of sudden death was met with, and judging from the more recent literature in cases which are carefully investigated sudden or unexpected death is extremely rare.

I am indebted to my former colleague, Dr. J. D. Rolleston, of the Grove Hospital, for permission to investigate cases under his care.

References.

(1) Fleming and Kennedy.—'Heart,' 1910-11, ii, p. 77.
(2) Hecht.—'Ergeb. d. inn. Med. u. Kinderheilk.,' 1913, xi, p. 324.
(3) Hume.—'Heart,' 1913–14, v, p. 25.
(4) Lewis, T.—'Clinical Disorders of the Heart-beat,' 2nd edition, 1913.
(5) Mackenzie, J.—'Diseases of the Heart,' 3rd edition, 1913.
(6) Magnus-Alsleben.—'Zeitschr. f. klin. Med.,' 1910, lxix, p. 82.
(7) Price and Mackenzie, Ivy.—'Heart,' 1911-12, iii, p. 233.
(8) Rohmer.—'Verhandl. der Gesselsch. f. Kinderheilk., Karlsruhe,' 1911, p. 279.
(9) Id.—'Zeitschr. f. exp. Path. u. Therapie,' 1912, xi, p. 426.
(10) Sperk und Hecht.—'Zentralb. f. Kinderheilk,' 1912, xvii, p. 309.
(11) Tanaka.—'Virch. Archiv.,' 1912, ccvii, p. 115.

SUBACUTE ATROPHY OF THE LIVER IN CHILDHOOD.

By R. A. Chisolm, M.A., M.D.Oxon, M.R.C.P.Lond.
Physician to the Queen's Hospital for Children.

Acute yellow atrophy, or, more correctly, acute diffuse necrosis of the liver, is a rare disease at any age, but is by no means unknown in childhood. Merkel in 1894 (10) collected eighteen cases and Schmidt (15) sixteen cases in 1897 in children of ten years of age and under, and other cases have been reported since then. The subacute form of the disease seems, however, to have attracted less attention. This is probably not due to the greater rarity of this type, for Milne (8, 9) in 1909 was able to record three cases that had come under his own observation, and in a second paper two years later alludes to two further cases that he had seen, and expresses his opinion that the subacute form of the disease is commoner in children than in later life. The more probable reason for the scanty number of cases that are to be found in the literature is twofold : in the first place the subacute form is less striking in the clinical picture it presents than is the acute type, and is therefore less likely to attract attention ; and, secondly, the symptoms and signs are, as will be

seen, uncertain in their significance and far from pathognomonic, and the diagnosis correspondingly difficult, and often only to be made as the result of autopsy. The disease, however, as seen in children presents several points of interest both in its clinical and pathological aspects, points which are illustrated by the following nine cases, which are all that I have been able to find after a fairly close search through the literature.

Case 1 (Venn) (16).—Girl, aged 7 years, good family history, and no previous illness. The child had been jaundiced for eight months previous to admission to the Victoria Hospital for Children in February, 1884, but had had no pain. There had been constipation and some vomiting after food, and the child had been quiet and drowsy. On admission the patient was a well-nourished child, deeply jaundiced. The abdomen was uniformly distended and tympanitic, but was not painful on pressure, and there was no ascites. The right lobe of the liver could be felt below the ribs, while the left lobe was uniformly enlarged, extending three fingers breadth below the ensiform cartilage. Bile was present in the urine, and the motions were light-coloured. The appetite was good, but there was great thirst. A few days after admission there was some hæmorrhage from the bowels and a hæmorrhagic eruption appeared on the body; there was also some bleeding from the gums. The liver at first became larger and then smaller; vomiting was severe during the last few days of life, and the patient became comatose and died about nine weeks after admission. At the autopsy there was no ascites. The liver weighed 300 grm. and was of yellowish-white colour; it was shrunken and the edges thinned; the surface was smooth. The right lobe was of nutmeg appearance with yellow infiltrations, and the left lobe showed yellow patches. The spleen was hard. Microscopically the liver cells were much atrophied, with granular *débris*. There was some evidence of interstitial hepatitis, but in no part had this proceeded to form fibrous tissue. There was an excess of bile capillaries, probably newly formed. No organisms could be seen, and the blood-vessels showed nothing noteworthy.

Note.—The diagnosis is given in the report of this case as acute yellow atrophy supervening on hypertrophic cirrhosis, but the presence of jaundice and the absence of excess of fibrous tissue renders it more probably an example of acute supervening on subacute atrophy, and as such I have included it here.

Case 2 (Klopstock) (5).—Girl, aged 10 years, who died of interstitial pneumonia, and who during life presented no symptoms of liver disease except a slightly enlarged liver. There was no jaundice. At the autopsy there was found typical subacute atrophy of the liver.

Case 3 (M'Donald and Milne) (1) (8).—Girl, aged 4 years, who had suffered from recurrent attacks of jaundice. The family history was good, and the child had previously suffered from chicken pox and measles. A fortnight before admission she was operated on for adenoids. Two years previously she had suffered from jaundice and was very languid, and complained of slight pains in the upper part of the abdomen. The attack lasted two months and was not attended by fever or other special symptom. Nine months later there was another attack of the same duration. Forty-two days before death jaundice appeared and increased in intensity. Bile pigment was present in the urine. The bowels were constipated and the motions pale; the temperature was regularly slightly subnormal. There was occasional dull pain and tenderness over the epigastrium; the weakness increased, but there was no other special symptom. There was no vomiting and the appetite was good. The child was admitted to the Royal Hospital for Sick Children under Dr. John Thomson five days after the jaundice developed. On admission the liver was somewhat enlarged, reaching 1 in.

below the costal margin. It felt smooth and rather firm; later the margin became slightly irregular. The spleen was also slightly enlarged. There was no ascites. On the twenty-third day of jaundice laparotomy was performed for possible gall-stones. The gall-bladder and bile-passages were healthy. The liver was not enlarged, but normal or slightly reduced in size. It was smooth and red-brown in colour. There were many bright green nodules on the surface of the size of a pin's head to a walnut, sharply defined and softer than the surrounding tissue, which was hard. The child died twenty-two days after the operation. At the autopsy the liver was slightly reduced in size and showed the same appearance as at the operation. Inside the organ were many well-defined green nodules, about half an inch in diameter. Microscopically these nodules showed extreme hyperplasia of the liver-cells. The nodules were ill-defined and irregular in outline. Some of the liver-cells were small, single nucleated and closely packed together; these were probably young newly-formed cells. Other cells were hypertrophic with two or more nuclei, and there were also giant liver-cells with thirty or more typical liver-cell nuclei, apparently an expression of regeneration. The tissue between the nodules was composed of young cellular fibrous tissue, with a few "bile-ducts." In some places the outlines of the destroyed lobules could be recognised. These were composed of dilated capillaries, generally more or less collapsed and overgrown by fibrous tissue, developed from the region of the portal tract. No elastic fibres could be seen in the fibrous tissue, such as are found in old-standing cirrhotic livers.

Case 4 (M'Donald and Milne) (2) (8).—Boy, aged 6 years. Thin, but well developed; previously quite healthy. He suffered from progressive jaundice and had got thinner and weaker for eight weeks. He died suddenly without the jaundice having become very intense and without any other special symptoms. He was only under observation for six days, during which the liver remained of normal size to percussion. At the autopsy the liver was uniformly slightly reduced in size; the surface was smooth and of a red-brown colour. Scattered irregularly over its surface were yellow areas of the size of a pin's head to 3 in. in diameter; they were slightly raised, fairly well defined, and of softer consistence than the surrounding fairly firm red-brown tissue. In the interior of the liver were irregular yellow masses, ½ to 1 in. in diameter in considerable numbers, some well defined, others with margins merging into the surrounding tissue. The microscopic appearances were very much the same as in the previous case, and the dilated capillaries were well marked, The authors say: "The appearances indicate very widespread, rapid destruction of the organ, and that compensatory regenerative changes had taken place by a multiplication of the preserved liver-cells and produced the yellow areas."

Case 5 (M'Donald and Milne) (3) (8).—Girl, aged 7 years. She had been out of sorts for two months, and for three and a half weeks had become progressively more jaundiced; she died in a state of delirium with high temperature. At the autopsy the liver was slightly enlarged and bile-stained. There were numerous small projecting yellowish nodules scattered irregularly through the organ and elevated above the surrounding pinkish-grey tissue. Inside the liver these areas were fairly well defined and separated by wide areas of pale pink-grey tissue. The yellow areas were composed of liver-cells in active proliferation, somewhat degenerated. There was marked evidence of hyperplasia; the intervening fibrous tissue was very young, and showed large numbers of bile-ducts.

Case 6 (Shennan, reported by M'Donald and Milne) (8).—Boy, aged 7 years. Till four years old he had been quite healthy; since then he had been getting gradually weaker. A year before death it was noticed that the abdomen was somewhat swollen; the ascites to which this was due had increased steadily in the last few months. For the last four months he had suffered from intermittent abdominal pain and diarrhœa. There was no obtainable history of jaundice, but during the three days he was in

hospital before he died there was an evident yellow colour of the sclerotics. Before death the liver was slightly enlarged. He died feverish, but with no increase of the slight jaundice. At the autopsy there was a considerable amount of fluid in the peritoneum and thorax. The mesenteric glands were enlarged and pale pink in colour; the spleen was congested, and the kidneys pale and showed cloudy swelling. The liver was somewhat enlarged, and weighed 930 grms.; it was greyish in colour with a slight yellow tint; irregularly scattered throughout the organ and projecting above its surface were numerous soft nodules of an orange-yellow colour. They varied in size from a pin's head to a small nut, and were sharply and regularly defined. Microscopically the isolated areas were composed of densely-packed liver-cells of very varying shape and size, and no lobular structure was to be observed. The intervening tissue was fibrous and not particularly dense in character, and contained very numerous irregular bile-ducts.

Case 7 (Dingwall Fordyce) (2).—Girl, aged 6 years 11 months. She had suffered from an attack of jaundice a year before, and since then from occasional pain in the left side of the abdomen. Seven weeks before admission she began to look ill and was kept in bed; since then the abdomen had become swollen, and pain was occasionally present. The rate of increase in the size of the abdomen had been greater for the two weeks before admission. Four days before admission the child was very tired and complained of headache, and next day the pain was worse, and there was vomiting with watery diarrhœa. The vomit then became bilious, and no motion was passed after the middle of the day before admission. There were no fits, but some twitching and screaming, and the child was comatose. On admission the nutrition was fair; the child was comatose with a dusky sunken face; there was no distinct jaundice, but the skin was sallow. There were œdema of the back, paresis of the left leg and the left side of the face, and a squint. The temperature was 99° F. The abdomen was very tense and distended; there was dulness in the flanks and a thrill. The liver and spleen were not palpable. A catheter specimen of urine showed a trace of albumin. At the autopsy there was a small amount of fluid in both pleura, and clear bile-stained fluid in the peritoneum. The spleen showed old-standing venous congestion. The liver was very reduced in size; the surface was grossly irregular, and showed well-marked yellow coloured nodules of ¼ to ½ in. in size, projecting above the greyish-pink substance of the organ. These yellow areas were clearly defined, and pervaded the interior of the organ. The substance between the yellow areas was greyish-pink in colour and very firm. Under the microscope the yellow areas showed liver-cells with a marked degree of hyperplasia; the grey tracts contained very few liver-cells, and were made up of fibrous tissue with numerous bile-ducts.

Case 8 (Rolleston) (14).—Boy, aged 4½ years. One other child was stillborn, and the mother had had two miscarriages. The patient suffered from jaundice for five weeks and ascites for two weeks before death, with hæmorrhages; 700 cc. of turbid ascitic fluid were withdrawn two days before the child died in coma. At the autopsy the liver weighed 375 grms.; the surface was irregular. On the left lobe was a raised area of the size of a florin. On section the liver was somewhat tough. Microscopically the appearances were those of acute yellow atrophy with regenerative changes in the area in the left lobe. There was also acute enteritis.

Case 9 (Porter Parkinson) (13).—Boy, aged 3 years. Admitted to hospital November the 19th, 1913. He had suffered from measles a year previously. Three weeks before admission there was a rigor followed by fever. Jaundice and drowsiness began three days before admission. On admission the boy was well-nourished; there was marked jaundice, and the stools were pale. The liver was enlarged, reaching 1 in. below the costal margin. The spleen was just palpable. The urine contained much bile, but no leucin or tyrosin. The temperature was normal, but rose to 103 on

November the 28th, and remained high for five days. On December the 12th there was friction at the base of the right lung, and the liver reached to the umbilicus, but then began to lessen in size and continued to do so till death. The jaundice persisted. On December the 28th a blood culture showed the presence of staphylococcus albus, which may, however, have been a contamination. There was some œdema of the hands and feet; there were no nervous symptoms. The temperature rose to 103 on December the 23rd, and continued high till death on December the 31st. Post-mortem the lungs showed numerous septic infarcts of the size of a pin's head. The peritoneum contained 170 cc. of yellow fluid. The liver was slightly enlarged and of pale brownish-yellow colour; there was plastic peritonitis round the portal fissure. On section the liver showed areas of a yellow colour, mingled with areas of reddish colour, sharply defined and hard. The spleen was pale, and contained one septic infarct. The kidneys were pale, with small septic infarcts. The abscesses in the lungs contained staphylococci, as did the vessels of the kidney. Under the microscope the red areas in the liver showed young connective tissue, with many dilated capillaries; scattered among these were bile-ducts, and some scattered liver-cells some of which were markedly hyperplastic and of the giant cell type, while some showed signs of degeneration. The yellow areas were composed of liver-cells in lobular arrangement, some showing hyperplasia, but many fatty and degenerating.

Taking the cases as a whole the justification for separating them as a group from the acute form of the disease, between which and cirrhosis of the liver they form a connecting link, is partly clinical and partly pathological; clinically the distinction lies in the greater duration of the case and the less violent nature of the symptoms; pathologically in the evidences of the attempts at repair that we find in the liver. As Milne (11) points out, both the subacute and the acute types start as an acute general necrosis of the liver parenchyma; in the latter the process is so severe that the patient dies before repair can be attempted; in the former the attempts at repair and regeneration of the liver parenchyma are evident in the hyperplasia of the liver-cells and the formation of new bile-ducts.

Of the nine cases here collected five were girls and four were boys, so that, unlike the acute form of the disease which is commoner in females than in males, there was no marked inequality of sex distribution, but of course the number of cases is very small.

Symptomatology.—Apart from Klopstock's case, which stands by itself in that the liver condition gave rise to no symptoms during life and was only discovered at autopsy, jaundice was present in every case and was usually the first symptom to attract attention. It varied in degree from the deep icterus of Case 1 to the evident yellow colour of the sclerotics of Case 6 and the sallow skin of Case 7, in which the presence of jaundice was only rendered certain by finding bile-stained fluid in the peritoneum after death. It is of interest that the changes in the liver in these two latter cases bore a nearer resemblance than did those seen in any of the other cases

in the series to the changes found in ordinary multilobular cirrhosis, in which disease jaundice of a marked degree is an uncommon event. On the other hand, in Case 1, in which the jaundice was deep, the liver picture was that of acute necrosis attacking a liver previously affected with subacute necrosis. It would appear, therefore, that the depth of the jaundice is more or less proportional to the acuteness of the changes in the liver. The jaundice when once established usually tended to become progressively deeper. In Cases 3 and 7 there was a history of previous attacks of jaundice, a sequence of events which has been recorded by Wentworth (17), and others in cases of the acute type of the disease in children. The possible significance of this history will be referred to later.

Ascites were present in Cases 6, 7 and 8. In two of these cases the changes in the liver were of the chronic type, approaching the cirrhotic, and the regenerative processes were widely diffused through the organ, while in the third case, No. 8, regeneration was limited to a small area in the left lobe, and the rest of the organ showed the microscopic picture of acute yellow atrophy. In none of the reports of these three cases is there mention of the condition of the portal vein or its branches.

The liver was somewhat enlarged at some period of the disease in all except Case 4, where it was normal to percussion, but slightly reduced in size at the autopsy, and Case 7, where it was not palpable during life and was very reduced in bulk after death. In Case 8 there is no note as to the size of the organ during life, but after death it was small. The enlargement was not always to be detected during life; it was usually regular, but in Case 1 the left lobe was more affected than the right, and in M'Donald and Milne's first case the margin, which was regular at first, later became somewhat irregular. Progressive diminution in size, which is such a marked feature in the acute type of the disease, and of considerable diagnostic importance, was noted in Cases 1 and 9, and in both cases the diminution followed a previous increase in size. In Case 3 the liver was enlarged to palpation on admission, but at the subsequent operation, eighteen days later, it was found to be normal or slightly reduced.

The spleen was noticed to be slightly enlarged in Case 3, just palpable in Case 9, and not palpable in Case 7. In the other cases there is no note of its condition during life.

Of the more general symptoms abdominal pain was of inconstant occurrence. Its absence is expressly recorded in Case 1, while in Cases 3, 6 and 7 it was present with intermissions, and was never

very severe in character. Vomiting was recorded in Case 1, in which it was severe, and also in Case 7, and was expressly stated to have been absent in Case 3. Light-coloured motions were the rule, but neither diarrhœa nor constipation were marked features of the series. Hæmorrhages, which are so notable a symptom in the acute form of the disease, were only seen in two of the cases, 1 and 8, in which, while regenerative changes were present in the liver, acute necrosis predominated. The temperature was noted as regularly and slightly subnormal in Case 3, high in Cases 5 and 6, normal in Case 7, and irregular in Case 9, in which, however, there was also present a general staphylococcal infection. The high temperature, therefore, which is usually seen in the acute type of the disease, falling towards the end, is often absent in the more chronic cases. Nor are the nervous symptoms nearly so pronounced in the latter as in the former.

Drowsiness with death in coma was seen in Case 1 and also in Case 7, while delirium was a terminal symptom in Case 5. Screaming and twitching, followed by coma with localised paresis of the face and leg on the left side, are recorded of Case 7, but nervous symptoms were expressly noted as absent in Case 9, and in none of the cases do they appear to have been very prominent.

In all cases where it is alluded to the urine contained bile ; leucin and tyrosin, so often found in the acute type of the disease, were noted as absent in Case 9, and in the other cases no mention is made of them.

Taking the symptoms as a whole there is a more or less regular gradation from cases such as 1 and 8, in which the picture closely resembles that seen in definite acute cases of the disease, with deep jaundice, hæmorrhages, severe vomiting (Case 1) and coma, and progressive diminution in the size of the liver, the chief sign of the subacute nature of the case being the long persistence of the jaundice, to cases such as Dingwall Fordyce's (2), marked by very slight jaundice, ascites and œdema, in which the clinical aspect of the case is very much that of ordinary multilobular cirrhosis of the liver.

A similar transition is seen in the pathological appearances. For instance, in Case 8 the only point in which the liver differed from the liver of typical acute yellow atrophy was in the presence in the left lobe of a small area showing regenerative changes. In Case 7, on the other hand, there were many areas showing regenerative hyperplasia with fibrous tissue intervening, and the microscopic picture was nearly that of a cirrhosis of the ordinary type, but the regenerative changes were more widespread and pronounced than is

usual in that disease. In Case 9, which, thanks to the courtesy of my colleague, Dr. Porter Parkinson, I had the opportunity of observing both during life and after death, necrotic and regenerative changes were closely intermingled. In places the necrotic changes had proceeded so far that the liver-cells had almost entirely disappeared, leaving a network of capillaries enclosing a few liver-cells of a degenerative type, while in other places regenerated liver-cells of the hyperplastic type were very prominent, though many even of these showed signs of necrosis. Clinically as well as pathologically the case occupied an intermediate position between the acute necrotic and the cirrhotic types of the disease. The regenerative changes have been studied among others by MacCallum (7), Muir (12) and Milne (11). The origin of the regenerated liver-cells is probably twofold; they may arise from pre-existing liver-cells or from the epithelium of the bile-ducts, which is gradually transformed into liver parenchyma. The tendency is for them to be collected together into masses of an ill-defined structure, resembling the adenomata, and it is probable that some of the cases reported as cirrhosis of the liver with adenoma formations are really examples of subacute atrophy. As to the ætiology of the disease, the present series of cases throws very little light on the point. Micrococci and bacteria have been found by Klebs (4) and by Dreschfeld (3) in acute cases of yellow atrophy of the liver, but their causal relation to the disease is at least uncertain. Cocci were present in the liver in Case 9, but as the patient was suffering from staphylococcal septicæmia their presence throws no light on the origin of the condition of the liver. The obstetric history of the patient's mother in Rolleston's case is somewhat suggestive of the presence of syphilis, but on the other hand in a series of sections from various parts of the liver of Case 9, stained by Levaditi's method, I failed to find a single spirochæte. Klopstock, in discussing the question in his case, is also against the syphilitic origin of the disease. Probably the condition is due to some acute infection the nature of which is at present entirely unknown.

A series of cases such as the present in which all the patients died, while valuable from the pathological point of view, obviously shed no light on the prognosis of the disease; but this is by no means to say that cases of subacute atrophy of the liver are invariably fatal. Indeed, Klopstock's patient died from a cause in no way connected with the condition of the liver, though that organ showed typical subacute atrophic changes. In the acute form of the disease there are several well-authenticated cases of recovery.

Wickham Legg (6) in 1880 collected twenty-eight cases of reputed recovery, and in two cases of the closely allied condition of malignant jaundice in pregnancy Creed and Scot-Skirving (1) reported two cases which recovered, both of which presented the classic signs of the disease, and in one of which leucin and tyrosin were found in the urine. Though therefore I can find no recorded cases of recovery in subacute atrophy, there seems but little doubt that such do occur; occasionally one meets children with cases of jaundice which at first appear to be merely of the catarrhal type, but in which the icterus persists longer than is usually the case in that complaint, while the constitutional symptoms are more pronounced. It seems legitimate to suppose that some at least of these are non-fatal cases of subacute atrophy. Further, the fact that certain of the cases in the series gave a history of previous attacks of jaundice raises the question whether such attacks were catarrhal or whether they were really instances of subacute atrophy which became quiescent but recurred in the final and fatal attack. Such attacks occurred in Cases 3, in which the history was peculiarly suggestive, and 7; in both the appearances found in the liver were not inconsistent with the possibility in question, for they obviously indicated a change of some duration. But if cases of subacute atrophy may recover, what is their ultimate fate? Possibly the liver may be restored to a normal condition, though this seems improbable; but when fibrous tissue is formed to the extent found for instance in Case 9 it is obvious that complete restoration to the normal cannot take place. Such fibrous tissue tends to supplant the more highly-developed liver parenchyma, and the ultimate picture would be one of a coarse, somewhat irregular, cirrhosis, such as that described by Marchand (9) under the name of multiple lobular hyperplasia, which in his case he regarded as the result of an attack of acute yellow atrophy six months previously. Now the ætiology of multi-lobular cirrhosis in children is obscure; alcohol is responsible for a certain number of cases, syphilis for some, and some have their origin in the acute infectious diseases, but I would suggest that a certain proportion of the remainder, for which no obvious cause can be found, take their rise from a previous attack of subacute atrophy of the liver.

As the disease presents a series of gradations between declared acute yellow atrophy of the liver and cirrhosis, the diagnosis must obviously be at times difficult or almost impossible. From the former the differentiation depends on the longer history and the less acute nature of the symptoms; the presence of ascites would

also be of value (Case 8), while the presence of jaundice of any considerable degree and the alteration in the size of the liver while the patient is under observation, both of which signs are unusual in cirrhosis, though the latter is more often seen in the juvenile than in the adult type of the disease, would help to differentiate the case from that condition. In the case which I had the advantage of watching, No. 9, the presence of jaundice, and the increase in size of the liver, followed by its diminution, rendered the diagnosis of subacute atrophy extremely probable, but the case both clinically and pathologically occupied an intermediate position, and there was therefore less likelihood of confusing it with either acute atrophy or cirrhosis. In cases where considerable ascites is present, differentiation from tuberculous peritonitis might cause a difficulty, but the presence of even a slight degree of jaundice would be against the latter disease, in which jaundice is a rare event. From catarrhal jaundice it is to be distinguished by the greater severity of the symptoms, the gradual progress downhill and the gradually increasing depth of the jaundice. In syphilitic cirrhosis of the liver jaundice is not a prominent feature, and the course of the case is very slow, while the presence of other signs of syphilis and of a positive Wassermann reaction would help to differentiate the case from one of subacute atrophy.

References.

(1) Creed, J. M., and Scot-Skirving, R.—'Austral. Med. Gaz.,' 1888–9, viii, p. 259.

(2) Dingwall Fordyce, A.—'Proc. Roy. Soc. Med.,' 1909-10, iii, Children's Sect., p. 1.

(3) Dreschfeld.—'Jour. of Anat. and Physiol.,' 1880-1, xv, p. 422.

(4) Klebs.—Quoted by Hunter, W., and Spriggs, E. I., 'Allbutt and Rolleston's System,' 1908, iv, pl. 1, p. 115.

(5) Klopstock, F.—'Virchow's Archiv,' 1908, cxcii, p. 254.

(6) Legg, Wickham.—'Bile, Jaundice, and Bilious Diseases,' 1880.

(7) MacCallum, W. G.—'Johns Hopkins Hosp. Rep.,' 1902, x, p. 379.

(8) M'Donald, S., and Milne, L. S., 'Journ. of Path. and Bact.,' 1909, xiii, p. 161.

(9) Marchand.—'Beitr. z. path. Anat. u. z. allg. Path.,' 1895, xvii, p. 206.

(10) Merkel, F.—'Münch med. Woch.,' 1894, xli, p. 86.

(11) Milne, L. S.—'Arch. Int. Med.,' 1911, viii, p. 639.

(12) Muir.—'Journ. of Path. and Bact.,' 1908, xii, p. 287.

(13) Porter Parkinson, J.—'Proc. Roy. Soc. Med.,' 1913-14, vii, Children's Sect., p. 74.

(14) Rolleston, H. D.—'Diseases of the Liver,' 1912, 2nd ed., p. 590.

(15) Schmidt.—'Dissertation, Kiel,' 1897.

(16) Venn, A.—'Lancet,' 1884, ii, p. 191.

(17) Wentworth, H. H.—'Arch. Pediatr.,' 1906, xxiii, p. 81.

FATAL HÆMOPTYSIS IN A CHILD AGED 4 YEARS.*

By J. D. Rolleston, M.D., and J. E. Robertson-Ross, L.M.S.S.A.,
Assistant Medical Officers, Grove Fever Hospital, London.

A boy, aged 4 years, was admitted to the Grove Fever Hospital on February the 19th, 1914, certified to be suffering from whooping cough. Measles, in December, 1912, was stated to be the only previous illness, but since November, 1913, he had appeared generally unwell and subject to attacks of abdominal pain and vomiting.

Condition on admission: The child was in a very ill-nourished and wasted state. The cough was paroxysmal, but at no time during his stay in hospital was he heard to whoop. Rhonchi and râles could be heard all over both lungs, but no areas of dulness could be marked out. The abdomen was distended and the superficial abdominal veins were dilated. The spleen could be palpated below the level of the costal margin and the area of liver dulness was slightly increased downwards. No enlargement of the lymphatic glands was detected. The temperature rose nightly to between 102° and 103°, but fell to nearly normal in the morning. The pulse-rate was rarely below 120 and the respiration varied 30 and 50.

On March the 15th a specimen of sputum was with difficulty obtained from the mouth, but no tubercle bacilli were seen. Von Pirquet's reaction, performed on April the 3rd, was also negative, a fact which is of interest in view of the subsequent post-mortem findings. (A positive reaction, however, was found in the patient's sister, aged 2 years, who was admitted on the same day with the certificate of whooping cough, and died on May the 21st of tuberculous meningitis—a terminal episode of what was probably a generalised infection. An autopsy could not be obtained, but three days before death some cloudy cerebro-spinal fluid was obtained on which Dr. Cartwright Wood, Bacteriologist to the Board, reported as follows: "We killed two guinea-pigs, inoculated with the cerebro-spinal fluid, and found both animals markedly affected with tuberculosis. We were able to demonstrate the presence of tubercle bacilli in the caseous material at the site of injection.")

A differential blood-count on April the 14th showed: Polymorphonuclears, 54 per cent., small mononuclears, 40 per cent., large mononuclears, 6 per cent. Very little change occurred in the child's general condition, but on April the 13th impairment of resonance

* A paper read at the Section for the Study of Disease in Children of the Royal Society of Medicine, on May the 22nd, 1914.

was noted below the left ninth rib behind and extending round into the left axilla; over this area the breathing was bronchial in character. During the evening of April the 24th a very little blood-stained sputum was expectorated, and at 8.30 p.m. on the 25th, after a comfortable day during which the child seemed rather brighter than usual, a sudden copious hæmoptysis occurred, death ensuing almost immediately.

Necropsy, April the 26th.—On pressing on the trachea and œsophagus a quantity of dark blood was expelled from the mouth and nostrils. The tracheo-bronchial glands were much enlarged, obviously tuberculous and some of them caseating. The left pleura was thickened and firmly adherent to the chest-wall and diaphragm. The upper lobe of the left lung was studded with tuberculous deposits. The lower lobe was solid, small pieces of it sinking in water, and presented a cavity about the size of a walnut, completely filled with recent blood-clot. The whole of the right lung was a mass of tuberculous nodules. The liver was enlarged, fatty and studded with tubercles. The spleen was considerably enlarged and contained tuberculous deposits of a fair size. Tubercles were also seen in the kidneys, basal meninges and grey matter of the cerebral cortex. The heart was empty and normal in size and appearance. The mesenteric glands were enlarged and the stomach contained a large quantity of altered blood but showed no sign of any lesion in the mucous membrane.

This case is of interest in the first place as being an example of general tuberculosis with the primary focus in the tracheo-bronchial glands producing a paroxysmal cough, simulating whooping cough, and in the second place owing to the rarity of hæmoptysis in so young a child. Although, as in seven out of twenty-three cases collected by Meusnier which came to autopsy the actual bleeding vessel was not definitely identified, it is presumable that the hæmoptysis occurred through the rupture of a branch of the pulmonary artery into the cavity found in the left lower lobe. Death was ascribed to asphyxiation rather than to the amount of blood lost.

A somewhat similar case occurred at the same hospital nearly three years previously. A boy, aged 4 years, was admitted with the certificate of whooping cough on June the 8th, 1911. His cough was sometimes paroxysmal, but no whoop occurred during his six months' stay in hospital. The signs and symptoms were those of chronic pulmonary tuberculosis, and the paroxysmal cough suggested affection of the tracheo-bronchial glands. Von Pirquet's reaction performed on July the 19th was positive. Two attacks of

profuse hæmoptysis occurred on December the 20th, and death took place on the same day. No autopsy could be obtained.

Meusnier (3) in 1892 collected thirty-three cases of hæmoptysis in children aged from 8 days to 14 years, but only sixteen of these were in children under 5 years. Of these sixteen all but three were tuberculous and all but one—a case of influenzal bronchitis—were fatal. The other two non-tuberculous cases occurred in new-born infants, in one of whom the lungs seem to have been digested by the inspiration of gastric juice during vomiting, while in the other the hæmoptysis was due to pulmonary thrombosis of syphilitic origin.

Magruder (2) in 1908 described a case in a negro boy, aged 3 years, very similar to our own, and referred to four other cases aged from 7 months to 3½ years, in which fatal hæmoptysis was due to pulmonary tuberculosis. Other cases of fatal hæmoptysis in children due to pulmonary tuberculosis have since been reported by Brown (1) in a girl of 3 years and by Thomas (5) in an infant of 3 months. It would thus appear that hæmoptysis occurring in young children is in the majority of cases due to pulmonary tuberculosis. It is as a rule a sudden and fatal event. In only five of Meusnier's twenty-nine fatal cases had the terminal hæmoptysis been preceded by any spitting of blood. In this respect the slight premonitory hæmoptysis which occurred in our case was exceptional.

The hæmorrhage may be caused by the rupture of an aneurysm or of a branch of the pulmonary artery into a cavity within the lung, either in the apex or the base, or by the breaking down of tracheo-bronchial glands surrounding a vessel and causing its rupture into a neighbouring bronchus. The fact that the mouth, pharynx and lower air-passages are as a rule filled with blood, demonstrates the mode of death in these cases. The stomach is generally found to contain much blood, suggesting at first sight hæmetemesis, but no lesions can be detected in the mucous membrane. Other causes of fatal hæmoptysis in children besides tuberculosis may be gangrene of the lung, ulceration of the neck-vessels in scarlet fever, or gangrenous angina or hæmorrhage in ulcerative sore throat, an example of which was reported by one of us to this section during the last session (4).

References.

(1) Brown, R.—'Lancet,' 1910, i, p. 1201.
(2) Magruder.—'Arch. of Pediat.,' 1908, xxiv, p. 519.
(3) Meusnier.—'Thèses de Paris,' 1891-2, No. 255.
(4) Rolleston, J. D.—'Brit. Journ. Child. Dis.,' 1913, x, p. 66.
(5) Thomas, E. H.—'Brit. Med. Jour.,' 1909, i, p. 1356.

Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, May the 22nd, 1914.

Mr. Sydney Stephenson, *Vice-President of the Section, in the Chair.*

Case of Mongolian Imbecility.—Dr. J. J. M. Shaw.—A boy, aged 3 years, born at full time. Labour was slow, and the child, which weighed 10 lb., was almost asphyxiated. The mother was aged 34 years when this her only child was born. There was a history of tuberculosis on the maternal side. Head brachycephalic, and fæces characteristic. Complexion florid, epicanthic folds large, and lips fissured. Little fingers incurvate, and toes, although presenting no abnormal cleft between the great toe and the next, unusually mobile. The child was very good-tempered and far from shy. His speech was very defective, and his code of gestures and strange noises were unintelligible except to his parents. Dentition did not commence until the age of 16 months, and his cry was that of an infant.

Spina Bifida Occulta.—Dr. Eric Bellingham Smith.—Boy, aged 9 years, was brought to the hospital for incontinence of urine. On examination the bladder was found to be distended as high as the umbilicus, and there was a constant dribbling of urine from the penis. The chief feature of interest, however, was the condition of the buttocks. These were flattened at the back, buttocks and posterior surfaces of the thigh presenting a practically level surface. The vertical cleft was entirely absent, and the anus was exposed and appeared to look directly backwards. There was a weak gluteal fold. The prominences of the trochanter and tuber ischii were well marked, and there was a cavity between them which was only filled by skin and fascia. Deep to this the deep external rotators of the thigh could be palpated, but the whole of the superficial gluteal muscles were absent. The muscles on the anterior and posterior aspects of the thigh were distinctly hypertrophied. The calf muscles seemed to be smaller than normal. This defect was, however, more apparent than real, and was probably produced by the marked hypertrophy of the thigh muscles above. The condition was absolutely symmetrical, and there was no affection of the trunk or arm muscles. The defect produced no disabilities. The child could run, walk and rise readily from any position in which he was placed. There had been no progressive wasting, and the condition had existed since birth. He had never exhibited any control of his urine since babyhood, and from time to time there had been incontinence of fæces. The reflexes were present and normal, and the mental condition was average for the age.

Congenital Defect, Sixth and Seventh Cranial Nerves.—Dr. Eric Bellingham Smith.—Boy, aged 3 years. Brought to hospital for weakness of one side of face. This defect had been noticed since second week of life. Delivery was moderately easy, and no instruments were used. There was complete seventh nerve paralysis on right side, and also paralysis of right external rectus. The condition suggested congenital defect of the sixth and seventh cranial nerves.

Osteoperiostitis of the Tubercle of the Tibia.—Mr. Chad Woodward.—Girl, aged 11 years, had complained of pain over the tubercle of the left tibia for six months; as she grew tired the pain became worse. There was an obvious swelling over the tubercle and tenderness was especially marked on the inner side.

Injury to the Deep Branch of the Ulnar Nerve.—Mr. Chad Woodward.—C. L., aged 15 years, fell on to a spike in June, 1913, and wounded his left wrist and palm. The wound was infected and healing occupied some weeks. There was paralysis of the interossei, ulnar lumbricales, and adductors of the thumb. Owing to the absence of sensory changes this lesion was prone to be overlooked at the time of the injury.

Old Injury to Elbow.—Mr. Chad Woodward.—Boy, aged 7 years, fell on a stone in August, 1913. Was treated with a splint for "fracture of a small bone and a bone out of place." All movements were now remarkably free, but the arm was very weak. The skiagram showed an anterior dislocation of the radius and the site of fracture of the ulna.

Case of Tremor.—Dr. F. Langmead.—Girl, 7½ years, with a peculiar tremor of the upper extremities. It was not associated with any evidence of disease in the nervous system or elsewhere. It was shock-like in character and brought on by volitional movement. It was first noticed when she was 7 months old, and there was no history of it having followed an acute illness. It was probably congenital. There was no record of it occurring in other members of the family.

Case of Abnormal Development and Fragility of the Bones.—Dr. Langmead.—Boy, aged 9 years 7 months, had always been backward mentally. He sat up at the age of 2 years 9 months, walked first when aged over 3 years, and was said to have begun to talk at the age of 1 year 6 months. His mother was afraid to let him out by himself as he was inclined to run without reason into traffic. He lay awake at night for several hours on end, but did not appear to be frightened and could not explain his wakefulness. He was extremely nervous. He had broken his arm on two occasions: when aged 4 and again when aged 7 years. The fractures were easily produced and occasioned by only very slight falls.

His limbs were long and thin, as also were his fingers and toes. Eyes somewhat staring and lids a little retracted. Both testicles had descended. Urine: Specific gravity 1008-1012, no albumen or sugar. No increased frequency of micturition. Pulse not easily felt; arteries not thickened. Blood-pressure 130 mm. Hg.

Specimen of Hydatid Cyst of the Brain.—Dr. C. Paget Lapage.—A boy, aged 10 years, after an injury to the head developed signs of cerebral tumour (optic neuritis, headache and vomiting) but no definite localising signs. A history of supposed tuberculous illness suggested that the tumour was tuberculous and definite improvement seemed to follow treatment with tuberclin. Later, localising signs—weakness and tremor of the right arm and leg and face—together with convulsions, developed. An exploratory operation was then performed. On incising the dura a large amount of clear fluid escaped; no brain matter could be seen, but the wall of a very large cyst presented at the opening. A small drainage tube was

left in, and through this large amounts of cyst-wall and clear fluid came away. Hernic cerebri and meningitis developed and death took place three weeks after the operation. No necropsy. Examination showed that the cyst was a hydatid.

Reference was made to the case reported by Henry Ashby in British Journal of Children's Diseases, 1904, i, *p.* 51.

The following papers were read:—**A Familial Case of Splenomegalic Anæmia with Infantilism.**—Dr. F. Parkes Weber (*vide* p. 345). **Nervous Cretinism.**—Major R. McCarrison, I.M.S. **Fatal Hæmoptysis in a Child, aged 4 Years.**—Dr. J. D. Rolleston and Mr. J. E. Robertson-Ross (*vide* p. 407).

CLINICAL SECTION.

Friday, May the 8th, 1914.

Spleno-myelogenic Leukæmia.—Dr. Clive Riviere.—Boy, aged 12 years, admitted to City of London Chest Hospital on December the 6th, 1913, with history of cough, breathlessness, pain in right shoulder, and general weakness of one year's duration. He then showed good nutrition with somewhat plethoric aspect, spleen to umbilicus, liver 1 in. below costal margin. Signs of enlarged thoracic glands, mainly consisting of increase of oval interspinous dulness on right side and impairment at lung apex. A skiagram confirmed this. Urine contained much albumen.

Blood-Count (December the 9th).

Red cells	2,920,000
Leucocytes	167,000
Hæmoglobin	55 per cent.
Colour index	0·9
Differential count:	
Polymorphonuclear neutrophiles	43·0 per cent.
,, basophiles	5·0 ,,
,, eosinophiles	4·0 ,,
Lymphocytes, large	1·5 ,,
,, small	2·5 ,,
Myelocytes, neutrophiles	42·5 ,,
,, eosinophiles	1·5 ,,

December the 13th. Put on liq. arsenicalis 3 mm., rising to 4 mm., and on December the 30th leucocytes had fallen to 137,000 per cubic millimetre.

On January the 9th, 1914, transferred to East London Children's Hospital so that X-ray treatment could be tried. After the first two doses a hard tube and aluminium filter were used.

Jan. the 14th	Leucocytes 328,000	Between January the 14th and February the 28th six X-ray doses of ½ B Sabouraud applied to the spleen.
,, the 16th	,, 279,000	
,, the 21st	,, 256,600	
Feb. the 5th	,, 362,200 (Myelocytes 56 per cent.)	

Feb. the 13th .	Leucocytes .	372,000	Between February the 6th and February the 27th X-rays in similar doses applied twice weekly to right and left leg alternately.
„ the 23rd .	„ .	310,000	
Mar. the 3rd .	„ .	334,000	
(A few nucleated reds seen for first time)			
Mar. the 16th .	Leucocytes .	349,200	Between March the 3rd and April the 20th benzol given in doses of 2 mm. rising to 6 mm. in petroleum emulsion.
Apr. the 20th .	„ .	400,800	
Apr. the 26th .	Leucocytes .	425,000	On April the 20th put back on liq. arsenicalis 3 mm., rising by April the 30th to 6 mm.

The red cells on January the 14th were 4,480,000 and the hæmoglobin 72 per cent., and they had remained much the same since. During the time the boy had been under observation he had felt well, had gained 6 lb. in weight, and his temperature had kept within normal limits. The pulse was accelerated, about 116. The liver had remained unaltered, but the spleen had increased in size both downwards and forwards. The area of lung impairment, indicative of large chest glands, had also increased, and now extended between the first and ninth dorsal spines on the right side. The last full differential leucocyte count obtained (April the 20th) was as follows:

Polymorphonuclear neutrophiles . .	28·5 per cent.
„ eosinophiles . .	2·0 „
Lymphocytes, large	3·5 „
„ small	3·5 „
Transitionals	12·5 „
Myelocytes, neutrophiles	39·0 „
„ eosinophiles	6·0 „

Two megaloblasts and one normoblast seen among 200 cells counted.

Philadelphia Pediatric Society.

May the 12th, 1914, William N. Bradley, M.D., President.

Spina Bifida.—Dr. James K. Young presented an eight months old child, cured by operation. The tumour was large and sessile, complicated by partial paralysis of both lower extremities and bilateral equinovarus. It was a multilocular meningocele, had ruptured at birth and was not infected. At six months, it was removed by dissection; the patient was suspended by the feet, two ounces of fluid were removed before operation, kept warm, and re-injected at the close of the operation. The sac was tied off by a through and through suture and purse-string suture, the wound protected by rubber dam attached by collodion. The patient was kept on the face for two weeks, enemata being given every five hours to secure evacuation of the bowels. Primary union was secured without infection and the baby was now well.

Dr. J. J. Hammond thought that surgeons must be interested in any operation that could cure spina bifida. He had recently observed a boy,

aged 10 years, under-weight and under-developed, with talipes equinovarus of the right foot, some muscular weakness of both legs and impaired function of the sphincters of bladder and rectum. The boy was of the type which tended to spontaneous cure, as no other would have survived so long and retained such a fair degree of development. The growth had a circumference of 3½ in., the greater part of which was indurated tissue overlying the actual cyst, which was about the size of a black walnut. The cleft involved the fourth and fifth lumbar vertebræ and proved to exist at the expense of the laminæ and spinous processes. Ample bony canal was exposed below the cyst, into which the cauda equina was replaced and retained without harmful pressure. The gap in the spine was closed by transplanting bone and the soft tissues brought together over it. Recovery was without incident, the boy leaving the hospital on the 21st day. The X-ray showed the transplanted bone united in perfect position, completely closing the cleft.

Dr. J. P. Crozer Griffith dwelt upon the diagnostic and prognostic indications of the different forms of spina bifida. The spinal meningocele, in which spinal meninges and fluid were contained, but without spinal cord, offered by far the best prognosis, lending itself most readily to operative interference. In the myelocystocele the cord was nearly always present, spread out upon and forming part of the wall of the tumour. The prognosis was grave, even after operation; even cases which survived operation were liable to retain evidence of paralysis. If he understood Dr. Young correctly, the central spinal canal communicated with the tumour in this case and this would put it in the class of myelocystocele.

Dr. Young stated that the child had double equinovarus. It was not a simple cyst, but a large multilocular cyst which had ruptured. He found an opening directly communicating with the spinal cord, which he was able to close. The mortality in these operations was about 87 per cent. in selected cases.

Some Points of Contact between Tuberculosis Work and Pædiatrics.—Dr. William Charles White, of Pittsburg, read this paper by invitation. He first referred to the problem which all branches of the medical profession must face in the growth of the paternalising governmental dominance of public health problems, and of the necessity for the various departments of the profession doing medical work for the public to correlate their forces and utilise these in the interest of the public welfare, so that when governments came to the point of complete care of the dependent and insured, they would find the medical profession far ahead of anything they could hope to attain. In this way the medical profession would retain its proper birthright—the care of the health of the people. Dr. White pointed out the well-known facts of the frequency of tuberculous infection in childhood, taking up the source of infection occurring from midwives during the first days of life, advocating a movement to procure medical examination of midwives, since they came into such intimate contact with the new-born. He spoke of the health of nurses, citing instances where the undoubted source of tuberculous infection in children could be traced to a nurse-girl or servant. Physicians must be sure that those to whom children were entrusted were not suffering from tuberculosis. He spoke of the common ground which pædiatrists and tuberculosis workers had in securing a municipally pasteurised milk supply; the relation of various infectious diseases as a groundwork on which the tubercle bacillus built its most virulent

infections; the lowered resistance to other diseases of children coming from tuberculous parents; the relation of tuberculosis to certain skin and eye conditions in childhood; and ended by urging that pædiatrists, tuberculosis workers and others interested in the public health should draw more closely the line of correlation which bound them together.

The Diagnosis of Chronic Tuberculosis in Early Life. — Dr. H. R. M. Landis did not consider acute tuberculosis, tuberculous meningitis, the acute broncho-pneumonia form following infectious diseases or surgical tuberculosis. The attitude of the various observers determined whether the recognition of tuberculosis in children was easy or difficult. Children of a tuberculous parent giving a positive tuberculin reaction or showing palpable cervical lymph-nodes had all been considered tubercular. Admitting that tuberculosis was commonly acquired in childhood, it by no means followed that this invariably occurred. Heredity, predisposition, race, poor food, bad housing, overwork, worry, illness, dissipation, and reinfection later in life, any of these might so lower resistance that tubercle bacilli, already implanted, began to multiply. Landis studied 362 children, brought to the Phipps Institute three or four years ago, because of tuberculosis in a parent. Within the past few months he had again observed 111 of these children. Over three years ago twenty-five were considered tubercular; all were now well except two, one of whom had died recently of tuberculosis, the other of an unknown cause. Slight daily elevation of temperature to between 99° and 100° F. was not abnormal in children. Physiologically normal physical signs were often exaggerated in children. Thus, sixty-seven of sixty-nine children with physical signs showed impairment and broncho-vesicular breathing at the right apex; yet, as Fetterolf had shown in adults, this was probably normal. Of the 111 children recently seen, seventeen had had a supposedly diseased right apex, and in two others both apices were said to have been affected; yet four years later seventeen of these nineteen were in good health, one still had a cough and only one had died of tuberculosis. The expired air might be directed into the pharyngeal vault, or against the roof of the mouth, simulating bronchial breathing all over the chest. Faulty posture also changed the breath-sounds. Since it was normal for children to show some enlargement of the lymph-nodes, it was not justifiable to consider enlarged cervical lymph-nodes an evidence of tuberculous infection. Physical signs supposedly due to enlarged bronchial glands were more fanciful than real, since the X-ray photographs taken of children with these signs had been uniformly negative. Dr. Landis advised against the wholesale massacre of tonsils simply because they were enlarged. The condition of the teeth was of importance in children, since bad teeth contributed to lower vital resistance. A positive tuberculin reaction did not mean clinical tuberculosis; nor did a negative test entirely shut out the possibility of tuberculosis. These tests were of no value except as an evidence of hypersensitiveness. The really important thing was to determine whether these children were physiologically normal; and if below par, the sociological evils were found to be the cause and not tuberculosis. If such a child continued living in insanitary surroundings it was sure to become infected in time. These cases were called pretuberculous. When removed from their bad surroundings they again return to normal. The main problem consisted in determining whether the child was physiologically normal; and if it was not, in making every endeavour to provide the means of making it normal.

Dr. W. S. Cornell spoke of the children in our public schools. No examination was possible, as the medical inspectors were not allowed to undress children. An attempt was now being made to group the supposedly tubercular children in open-air classes. There were now one open-air class and seven classes held in rooms with open windows. It was almost impossible to decide whether most of these children had tuberculosis or not.

Dr. S. McC. Hamill said that work undertaken with Drs. Carpenter and Cope to determine the incidence of tuberculosis in children at St. Vincent's Home had emphasised three points: that the conjunctival test was not without danger, that it was much more disturbing in its application and results than the Von Pirquet and More tests, and that it was not any more or any less sensitive than those. They advised that the eye-test should not be used for diagnosis in children. Dr. Hamill believed the routine application of the Von Pirquet test advisable, since a positive result probably indicated the existence of a focus of tuberculosis. This should stimulate us to give great attention to the hygiene of such children. Dr. Hamill had found the tuberculin test very confusing when positive in children having pulmonary lesions, indefinite febrile conditions or some chronic impairment of health. He referred to a case, a baby, aged 11 months, brought to the Polyclinic Hospital Dispensary by a mother, with advanced tuberculosis. The baby was emaciated, extremely anæmic, having broncho-pneumonia. It was so nearly moribund that it was placed in isolation, with the expectation that it would die within forty-eight hours. The original diagnosis of tuberculosis was confirmed by a positive Von Pirquet test. The infant did not die, but was sent out at the end of six weeks, with lungs normal and nutrition good. Either the entire process was tuberculous and the child recovered, or the positive tuberculin tests indicated a limited focus of tuberculosis and the child's serious illness was due to non-tuberculous broncho-pneumonia.

Dr. J. P. Crozer Griffith said that numbers of autopsy statistics showed tuberculosis to be a very common affection of early life. But it was certainly often very difficult to make the clinical diagnosis. This was true also in the acute cases. In infancy the diagnosis between a neurotic state due to other causes and that dependent upon general tuberculosis could often not be made until autopsy; and there was yet no method known to distinguish with certainty between simple and tuberculous broncho-pneumonia, except upon discovery of tubercle bacilli in the secretion, which was by no means always easy. In older children acute miliary tuberculosis simulated typhoid fever until, perhaps, tuberculous meningitis appeared. Outside of the joints, meninges and peritoneum, tuberculosis was not frequent in children, at least clinically. Small, scattered lung foci could not be discovered by clinical examination and passed into tuberculous broncho-pneumonia; and chronic phthisis was very rare. This was not against the well-known frequency of tuberculosis in early life, but against the power to recognise it. He was very sceptical regarding the many published lists of cases attempting to show by clinical examination the frequency of tuberculosis at school-age.

Société de Pédiatrie, Paris.

May the 12th, 1914. Bulletin No. 8.

Chronic Appendicitis and Inguinal Hernia.—M. Veau had collected fifty-three cases of inguinal hernia operated on by him. In twenty-four simple cases the appendicitis was overlooked in one half. He was of opinion that when a child, the subject of inguinal hernia, complained of pain, appendicitis should be suspected. A normal inguinal hernia was not painful. The appendix should be systematically examined in every child who has a hernia. Ablation of the appendix by inguinal incision was possible in young infants; later an incision over McBurney's point was necessary.

Heliotherapy in Surgical Tuberculosis.—M. Trèves gave an account of his experiences at Leysin and on the French Mediterranean littoral and insisted on the immense benefit obtained by a sea cure combined with heliotherapy. He recommended exposure of the whole body to the sun during several hours each day, whether the joints were immobilised or not. The cure should not supersede the surgical treatment of sequestræ.

Generalised Sarcoma in an Infant.—MM. Hallé and Françon showed pathological specimens of a boy of 3 months, admitted for intense dyspnœa and fever (40·4°). There were also marked œdema of the lower part of the body, numerous tumours on the skin and exophthalmos.

Two Cases of Cerebro-spinal Meningitis cured by Injections of Anti-parameningococcal Serum after Failure with Anti-meningococcal Serum.—MM. Delort and Stiassnie related the cases of two children, aged 4 and 7 months, who presented all the symptoms of cerebro-spinal meningitis, and in whom a diplococcus analogous to the meningococcus was found.

Purpuric Form of Pneumococcal Septicæmia.—MM. Rolland and Buc reported the case of a child of 15 months admitted to hospital with fever and suppurative otitis. He subsequently developed purpura and meningitis, of which he died. A blood-culture taken during life showed the presence of the pneumococcus in the pus of the ear, the cerebro-spinal fluid and the serum of the purpuric skin.

Study of the Blood-dust in Infants.—MM. Nobécourt and Maillet found that in infants whose digestion was good the blood-dust was abundant, while the amount diminished or even disappeared in the ill-nourished or those on a water diet. In general the amount of blood-dust was in relation to the richness of the milk in fat.

Azœtæmia and Fæcal Ash in Athreptics.—MM. Nobécourt and Bidot, taking into consideration the loss of mineral matter which plays so important a part in Parrot's athrepsia, had made researches as to the amount of urea in the cerebro-spinal fluid and the relations between the amount of

ash in the fæces to that in the food. The quantity of fæcal ash was always less than the amount ingested. In athreptics, who are azœtæmic in proportion to the cachexia, the proportion of ingested ash was 12 to 25 per cent., while it reached 28 to 30 per cent. in those cases which were on the way to recovery.

Hydatid Cyst in the Brain of a Girl of fourteen years.—MM. Cassoute and Bocca. A woodcut of the post-mortem appearances is given in the 'Bulletin.' There were no physical signs to explain the occurrence of epileptiform attacks.

Benign Encephalitis and Acute Ataxy.—MM. Guinon and Aine related the case of a boy of 4 years who, after an attack of measles, became suddenly unconscious. A phase of agitation and choreiform movements followed lasting ten days. In a second case the onset was that of a cerebro-spinal meningitis with unconsciousness, torpor, and then general rigidity. Recovery took place after two months.

Cyst in the Glosso-epiglottic Fold.—MM. Abraud and Masson described a case which resembled ranula in its histological structure.

Polypus of Pharynx.—M. Abraud showed a large fibro-mucous polypus the size of a hen's egg which he had removed from the pharynx of a child of 11 years.

Acute Renal Insufficiency during latest Suprarenal Tuberculosis.—MM. Méres and Henyer reported the case of a boy aged 14½ years admitted to hospital in a state of prostration and unconsciousness with a temperature of 104° F. and pulse of 140. Serum diagnosis and lumbar puncture gave negative results. Autopsy showed lesions of enteric fever and tuberculosis of the suprarenal capsules.

Congenital Syphilis with Inflammation of both Tibiæ.—MM. Méres and Henyer reported the case of a boy, aged 14 years, admitted into hospital complaining of pains in the legs, with slight adenopathy and rise of temperature. Radioscopy showed periosteal thickening of both tibiæ.

Unilateral Glaucoma during Tuberculous Meningitis.—MM. Jules Renault and Thiers showed pathological specimens from an infant who, during an attack of ordinary tuberculous meningitis, had a marked prominence of the left eye associated with hypertension of the eyeball and considerable dilatation of the pupil on the same side. A mass of tubercle the size of a nut occupied that part of the floor of the fourth ventricle which corresponded to the nuclei of the fourth and sixth and seventh nerves.

Post-diphtheritic Laryngo-tracheal Cicatricial Stenosis.—MM. Savariand and Zaepffel reported a case in a boy, aged 4 years, cured by laryngostomy.

Vincent Dickinson.

The Fifth International Congress on School Hygiene.

This Congress will be held in Brussels in 1915. The programme contains the following subjects: (1) "The School Building and its Equipment," (2) "Medical Inspection in Urban and Rural Schools," (3) "The Prevention of Contagious Diseases in Schools," (4) "Teaching of Hygiene to Teachers, Scholars, and Parents," (5) "School Hygiene in Relation to Physical Education at Different Ages," (6) "Teaching Methods, Syllabusses, and School Equipment in Relation to Hygiene," (7) "School Hygiene in Relation to Exceptional Children," (8) "School Hygiene in Relation to Adolescents."

This Congress is placed under the patronage of H.M. King Albert of the Belgians and under the auspices of the Belgian National Institute of Pædology and of the Belgian Pædotechnical Society; the organising committee is presided over by Mr. J. Corman, Director-General of the Primary Studies at the Department of Science and Art, and of Dr. J. Demoor, rector of the Free University of Brussels.

All communications and inquiries should be addressed to the Secretary, Dr. H. Rulot, 66, Rue des Rentiers, Brussels.

Abstracts from Current Literature.

Medicine.

Protection of the infant against tuberculosis in the first year of life (*'Arch. de Méd. des enf.,'* 1914, xvii, *p.* 241).—**P. Nobécourt** and **G. Schreiber** find that: (1) the infant is not born tuberculous and that only exceptionally is tuberculosis hereditary. It is nearly always contracted by contagion after birth, and the contagion is always familial; (2) tuberculosis of the child is then inevitable, and the only means of preventing contagion is removal to a healthy milieu; (3) the proportion of tuberculous children increases rapidly with age, and in order to be effective removal from the infective source to the country or seaside should be as soon after birth as possible. F. R. B. Atkinson.

Latent tuberculosis in infants (*'Monatsschr. f. Kinderheilk.,'* 1914, xii, *p.* 618).—**T. A. Ossinen** examined 100 bodies of children, 55 males, 45 females, as far as the peritracheal, peribronchial, and mesenteric glands were concerned by antiformin, histological examination and inoculation into guinea-pigs. In ninety-seven, microscopically nothing was found, and examination with antiformin and by inoculation gave a negative result; in the three other cases microscopically the examination was negative but positive by the other two methods. F. R. B. Atkinson.

A study of the child in the tuberculous milieu (*'Arch. of Ped.,'* 1914, xxxi, *p.* 96).—**M. Fishberg** investigated 217 families, and found among 1129 persons, 792 under 15 years of age. Nearly all were reared on breast-milk, only 5·5 per cent. had been artificially fed. Infants suckled by consumptive mothers thrived as well as others of their class, but many became infected. The weight of the infants was much short of the normal. The form of the chest was normal in 62·5 per cent., 27·5 had flat chests, and 10 per cent. had

rachitic chests. In 8 per cent. enlarged superficial thoracic veins were found. The cervical glands were swollen in 67·8 per cent. Enlarged tonsils, adenoids, etc. were found in 58·6 per cent. The cutaneous tuberculin test was positive in 7 per cent. of infants between one and six months. At 14 years 53·79 per cent. were found infected with tuberculosis. The tuberculous milieu did not materially increase the number infected with tuberculosis among the children over 6 years of age, but the proportion was much higher under 4 years. The prognosis depended largely on the age at which infection took place, the danger of tuberculosis as a fatal disease among children, was in an inverse ratio to the age at which they were infected. Those infected during the first two years of life were in grave danger of succumbing to hæmatogenous tuberculosis, especially tuberculous meningitis.

F. R. B. Atkinson.

Chronic tuberculosis of the peritoneum in infancy (*'Le Nourisson,'* 1914, II, *p.* 130).—**A. B. Marfan.**—Chronic tuberculosis of the peritoneum is commonest between six and fifteen years of age, rare before two. In infancy ascitic and fibro-caseous forms occur. The affection is much graver than in older children. The author differentiates the large flaccid abdomen from the large tympanitic one; the former is associated with symptoms of slight dyspepsia, the latter may be transitory, intermittent and limited to the region of the stomach, or intermittent and generalised. In the large tympanitic abdomen the disease may be chronic with diarrhœa and due to intestinal ulcerations, chronic with constipation, and chronic without diarrhœa and without constipation, and this the author believes is ordinarily due to tuberculosis of the peritoneum or mesenteric glands, without affection of the intestine.

F. R. B. Atkinson.

Hæmatology of infantile tuberculosis (*'Gaz. internaz. di med. chir., etc.,'* 1914, *pp.* 409 and 448).—**S. Maggiore,** as a result of observations on children of all ages and suffering from various forms of tuberculosis, comes to the following conclusions: (1) There is no single and constant hæmatological formula for any stages or form of tuberculosis. Variations occur in every case. (2) From cases with a normal finding one passes to those with reduction of the hæmoglobin and red cells until in exceptional cases, especially in infants, a high degree is reached. (3) The white cells are only exceptionally diminished; as a rule they are normal or increased. Hyperleucocytosis, however, is rare. (4) Both in infants and older children there is always an increase in the number of mononuclears and transitionals. This increase affects the lymphocytes, only exceptionally the polymorphs. (5) The basophiles in most cases are deficient or behave as under normal conditions. (6) The eosinophiles in exceptional cases are increased; as a rule they are normal or diminished. (7) The appearance of anomalous forms is not frequent. When there is a marked reduction of hæmoglobin and of the red cells, polychromatopsia, anisocytosis, and exceptionally myelocytes may be found.

J. D. Rolleston.

Tuberculous bacillæmia (*'Osp. dei Bamb. di Milan,'* 1913, II, *No.* 9).—**L. Pollini** examined the blood of adults in an advanced stage of pulmonary tuberculosis and of children affected with acute miliary tuberculosis, acute and chronic pulmonary forms and various forms of surgical tuberculosis. As in thirty-eight cases direct microscopic examination never showed any bacillæmia, and in sixty-five cases the blood of tuberculous patients never

infected guinea-pigs, Pollini concludes that tuberculous bacillæmia is extremely rare, at least in the common forms of tuberculosis. He attributes its alleged frequency to mistaking the acid-fast bacilli present in ordinary distilled water for tubercle bacilli. J. D. Rolleston.

The bitonal cough in tuberculosis of the glands of the trachea and bronchi in infants (*'Le Nourisson,'* 1913, I, *p.* 325).—**A. B. Marfan** and **D. Mantoux.**—Marfan has noticed this peculiarity of the cough in this condition in children less than two years of age, and considers it a valuable sign. There are two distinct sounds in the cough, the first low and veiled and the second higher and almost singing. In ten cases that came to autopsy and which during life had this cough eight showed the above lesions. It indicates a severe and serious condition of the glands.
F. R. B. Atkinson.

The ætiology of orthotic albuminuria, with especial reference to its relationship to tuberculosis (*'Arch. f. Kinderheilk.,'* 1913, LXII, *p.* 34).—**F. Wendenburg** considers that infection plays a part in the production of this form of albuminuria. As regards tuberculosis, he found that of 250 children from nine to thirteen years of age orthototic albuminuria was present in 37 per cent. of cases which showed a positive von Pirquet reaction. F. R. B. Atkinson.

Scrofuloderma in the first year of life, and the results of treatment in tuberculosis (*'Monatsschr. f. Kinderheilk.,'* 1914, XII, *p.* 699).—**K. Bahr** describes five cases, in only one of which tuberculin was used. The other four recovered permanently without treatment, and there were no signs of tuberculosis elsewhere after an interval of two years. The cases show that in the first year of life scrofuloderma is often very mild, and easily cured. F. R. B. Atkinson.

Disseminated miliary tuberculosis of lungs and skin (*'Amer. Journ. Dis. Child.,'* 1914, VII, *p.* 24).—**W. P. Northrup** reports eight cases in children, aged from 15 months to 9 years, illustrating the diagnostic value of X-rays and tuberculous eruptions. The cases presented the mental pictures of pneumonia, but no physical signs of bronchitis. The skin lesions also possess prognostic value, as their presence is almost always associated with a fatal form of tuberculosis in the young child. The individual lesions are of the size of the typhoid rose-spots. They are topped by a tiny vesicle, surrounded sooner or later by a congested or hæmorrhagic zone, with the formation of a crust, which, when removed, leaves a little pit. Diagnosis must be made from chicken-pox, molluscum contagiosum, and syphilis.
J. D. Rolleston.

Radiographic studies of the chest in tuberculous meningitis (*'Arch. of Ped.,'* 1913, XXX, *p.* 659).—**I. O. Woodruff** bases his report on fifteen cases of tuberculous meningitis, and finds that there is usually considerable pulmonary involvement in tuberculous meningitis, and that when the former exists the radiographic shadows can be relied on to diagnose the pathological process, and hence can aid in the diagnosis of meningitis.
F. R. B. Atkinson.

Transitory attacks of tuberculous meningitis (*'Bull. et Mém. Soc. Méd. Hôp. de Paris,'* 1914, XXXVII, *p.* 252).—**H. Barbier.**—A boy, aged eight years, subject to repeated attacks of bronchitis, was admitted to

hospital in November, 1911, with pleural effusion and sputum containing tubercle bacilli. The effusion gradually cleared up, the tubercle bacilli disappeared, and the sputum ceased. At the beginning of March, 1912, meningeal symptoms, consisting of headache and vomiting, occurred, and lasted for a week, together with signs of active disease in both lungs. On April 10th the meningeal symptoms returned, and a fortnight later intercostal herpes developed, accompanied by exaggerated reflexes, ankle clonus, unequal pupils, and irregular pulse. The cerebro-spinal fluid was normal. Tubercle bacilli were found again in the sputum. The herpes and meningeal symptoms disappeared by May 5th, and the signs in the lungs became less marked. Vomiting, headache, and exaggeration of the right knee-jerk occurred again from May 23rd to 26th and from June 11th to 15th. Subsequently improvement occurred until January, 1914, when the boy showed active disease of the lungs and exaggeration of the knee and ankle jerks, which Barbier regarded as a permanent stigma of the meningeal episode in April, 1912. He had reported similar cases before (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1912, IX, *pp.* 178 and 278).

J. D. ROLLESTON.

Cerebellar ataxia in tuberculous meningitis ('*Arch. de méd. des enf.*,' 1914, XVII, *p.* 367).—**A. d'Espine,** who recently drew attention to disturbance of equilibrium in tuberculous meningitis (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1913, X, *p.* 513), records a case of a boy, aged $2\frac{1}{2}$ years, in whom cerebellar ataxia was an early sign of tuberculous meningitis. In addition to the ordinary post-mortem findings, a solitary tubercle was found in the posterior part of the superior vermis.

J. D. ROLLESTON.

Action in vitro of extracts of lymphatic glands, and various normal organs on the tubercle bacillus ('*Journ. de Phys. et de Path. Gén.*,' 1913, XV, *p.* 835).—**A. B. Marfan, B. Weill-Hallé** and **H. Lemaire** find that the lymph-glands and normal spleen attenuate the virulence of the tubercle bacilli. The tissue of the liver has a similar but less marked action. The serum does not possess this action. The cerebral pulp on the contrary increases the virulence of the tubercle bacillus. It cannot be determined whether the product which possesses this attenuating action is of the nature of an enzyme or lipoid.

F. R. B. ATKINSON.

The passage of Koch's bacillus in the milk of tuberculous wet-nurses ('*Journ. de Méd. de Bordeaux*,' 1914, LXXXV, *p.* 93).—**B. Auché** states that animals injected with milk from tuberculous wet-nurses had never become tuberculous in the experience of numerous authors. In a third of the cases, however, others have found that infection takes place. By employing antiformin tubercle bacilli had nearly always been found in the milk.

F. R. B. ATKINSON.

Muscular work and bodily constitution ('*Arch. f. Kinderheilk.*,' 1913, '*Baginsky Festschrift*,' *p.* 515).—**H. Roeder** finds that muscular work can influence bodily constitution to a high degree, and excites growth and weight. Hence in young persons movements in the open air, together with good food and hygienic conditions, are essential, especially as prophylactics against tuberculosis.

F. R. B. ATKINSON.

Tuberculosis and the von Pirquet test in children ('*Med. Record*,' 1913, LXXXIV, *p.* 1032).—**L. Shalet** considers that no ambulant cases of

possible tuberculosis occurring under 16 years of age should be diagnosed as such unless the von Pirquet test is positive. He also thinks that no child is suffering from tuberculous infection who does not react to the test, unless he is too ill to react or is suffering from some intercurrent acute infection. F. R. B. Atkinson.

Von Pirquet's reaction in children (*'Jahrb. f. Kinderheilk.,'* 1914, lxxix, *p.* 71).—**I. Cronquist** found that the size of the papules varied considerably. They were much larger in children with an exudative diathesis than in others. The individual papules also varied in size with five vaccinations in the same children, whereas in those of a non-exudative diathesis the five papules were of the same size. The tuberculin reaction was stronger if the dilutions were made with water than with physiological saline solution, and two vaccinations were made, one with undiluted old tuberculin and one with Beranek's tuberculin. The papule which arose at the site of injection of old tuberculin only was larger than when no Beranek's serum was used. F. R. B. Atkinson.

Tuberculin skin reactions in infancy (*'Arch of Ped.,'* 1913, xxx, *p.* 665).—**A. Brown** found that seventy of the hospital cases under two years giving a positive reaction proved fatal, and the lesions were normally general in distribution. If the first test be negative, the test should be repeated if tuberculosis be suspected. A negative cutaneous reaction in infancy, except in moribund cases or in children suffering from measles, is almost certain evidence against tuberculosis. Six hundred and fifty cases were tested. F. R. B. Atkinson.

The diagnostic and prognostic values of repeated local tuberculin reactions, with remarks on the meaning of tuberculin hypersensitiveness (*'Jahrb. f. Kinderheilk.,'* 1914, lxxix, *p.* 123).—**G. Bessan** and **J. Schwenke** used three dilutions of Koch's old tuberculin 1 : 10,000, 1 : 1000, and 1 : 100. The dilutions were made every eight days. An intracutaneous injection of 0·1 cm. of the weakest solution was given on the extensor side of the thigh, and the extent of the reaction noted. A second injection was given eight days later at the same spot on the other thigh. If the result was positive the same solution was repeated; if negative the next strongest, and so on. It was found that clinical cases of tuberculosis showed no increase of local tuberculin sensibility. In children a strong local reaction usually indicated an active process. Feeble reaction pointed to a progressive or inactive condition. Marked increase of local sensibility excluded active disease. There was a quantitative and qualitative difference between the courses of local tuberculin reactions and serum hypersensitiveness. F. R. B. Atkinson.

Experiences with Rosenbach's tuberculin in the treatment of internal tuberculosis in children (*'Arch. f. Kinderheilk.,'* 1914, lxii, *p.* 337).—**A. Stommel** is not favourably impressed with this tuberculin. Of eighteen cases treated with it for tuberculosis of the lungs and bronchial glands six were markedly improved, four showed slight improvement, five no improvement, three became worse and two of these died. Disappearance of the lung condition occurred in only one case; there was no anti-pyretic action. In three cases of tuberculous peritonitis improvement occurred. F. R. B. Atkinson.

The significance of chronic cough and wasting in childhood (*'Clin. Journ.'*, 1913, XLII, *p.* 437).—**E. B. Smith** limits himself to children between three and thirteen suffering from these symptoms, which he finds are chiefly associated with some intestinal derangement. The cough is dry and hacking, and is noticed on first going to bed or rising in the morning. The throat is usually congested and adenoids are frequently present. Wasting may be slight or excessive, and there is often pain in the epigastrium. The appetite is bad; constipation alone or alternating with diarrhœa is frequent. Pallor, cold hands and feet, sweating, most marked at night, flabby muscles and atony of the bowels are common. Sodæ bicarb. tr., rhei co. and tr. nucis vom., are good remedies. The teeth should be carefully attended to. Meals should be regular; sweets should be avoided. These cases are commonly classed as tuberculous, especially tuberculous bronchial glands. F. R. B. Atkinson.

Respiratory infections in infants' wards (*'Amer. Journ. Dis. Child.,'* 1914, VII, *p.* 380).—**W. F. Chappel** and **A. Brown** during the past two years had 129 cases of acute naso-pharyngeal infection in the infants' wards, and not a single case of measles, whooping-cough, or scarlet fever. During the same period, out of 271 marasmus babies only 29 died from uncomplicated marasmus, while of the total deaths (97), two-thirds were due to complications chiefly respiratory. Pneumococcus and streptococcus infections caused most of the trouble. As prophylactic treatment they recommend: (1) increasing the child's resistance by careful attention to its nutrition; (2) careful nursing to minimise danger of transmitting infection; (3) direct prevention of respiratory infection by post-nasal douching. Warm antiseptic solutions were used, chiefly boracic acid, followed by argyrol solution in various strengths. J. D. Rolleston.

Dorsal percussion in enlargement of the tracheo-bronchial glands (*'Ann. Journ. Med. Sci.,'* 1913, CXLVI, *p.* 660).—**J. C. da Costa, Jun.**—Of eighteen cases of tracheo-bronchial glandular affection two-thirds showed interscapular dulness above the level of the inferior scapular angles, or over the first seven thoracic spinous processes; two dulness below the inferior scapular angle, three general thoracic hyperresonance over the entire thoracic segment, and in one there was no impairment of resonance below the level of the third thoracic spine. He believes that in simple enlargement of the tracheo-bronchial lymphatic glands percussion of the thoracic vertebræ, especially above the level of the inferior scapular angles, usually affords tonal changes of real value. F. R. B. Atkinson.

Perforation of a bronchus by a glandular abscess (*'Riv. Osped.,'* 1913, III, *p.* 668).—**U. Mancini.**—A boy, aged 3 years, who had always been delicate, developed a paroxysmal cough resembling pertussis, in convalescence from measles. The cough persisted for about a year, and was accompanied by irregular fever. The general condition had been getting worse for a week before admission, and the fever had been high. Death took place from suffocation following an attack of coughing two days after admission. Post mortem there was a diffuse adenopathy of the bronchial tracheal and aortic glands, and the right bronchus was found to be perforated by a glandular abscess in which were a very few bacilli and isolated cocci. There were also enlargement of the liver, spleen and mesenteric glands and

follicular colitis of the last segment of the colon. Reference is made to the similar case reported by Cacciami (*vide* British Journal of Children's Diseases, 1912, ix, *p.* 464). J. D. Rolleston.

Subcutaneous emphysema in children ('*Arch. de med. des enf.*,' 1914, xvii, *p.* 81).—**J. Comby.**—This may be due to a lesion of the nasal fossæ, facial sinuses, larynx or trachea (intubation or tracheotomy), bronchi and lungs (foreign bodies, thoracentesis, rupture of alveoli, or glandular or pulmonary cavities). It is rarely due to a lesion of the œsophagus, stomach or intestine. Glandulo-pulmonary tuberculosis is a frequent cause and then in order of frequency spasmodic bronchitis, pertussis, broncho-pneumonia, pneumonia, diphtheria and dilatation of the bronchi. The air penetrates into the cellular tissue of the mediastinum, reaches the base of the neck and gradually extends over the face, trunk and limbs. Dyspnœa and cyanosis are almost always present. The emphysema may disappear spontaneously in one or two weeks. The prognosis is bad in half the cases. The dyspnœa should be treated with inhalations of oxygen, intestinal derivatives, antispasmodics, digitalis and theobromine, and warmth to the extremities. Comby records 10 personal cases in which the emphysema was due to bronchitis, broncho-pneumonia, pulmonary tuberculosis, thoracentesis and measles without broncho-pneumonia. J. D. Rolleston.

The prognosis of generalised emphysema in children ('*Gaz. des Hôp.*,' 1913, lxxxvi, *p.* 1901).—**Rivet** reports two cases. The first was a child, aged 7½ months, in whom the emphysema was the terminal event in double broncho-pneumonia; the second was a child, aged 4 years, in whom it supervened in the course of slight bronchitis, and cleared up in a week. The prognosis varies greatly, it is worse the younger the child, and when there is severe lung disease and a general toxic state. The treatment consists in checking the cough, wet compresses to the thorax, and oxygen inhalations. The induction of pneumothorax is impracticable, as one cannot discover in which lung is the lesion causing the emphysema.

J. Porter Parkinson.

Malarial asthma ('*Arch. Brasil. de med.*,' 1913, iii, *p.* 776).—**T. de Almeida, Jun.**, records a case in a girl, aged 7 years. The ordinary treatment for asthma proving ineffective, examination of the blood was made and showed the presence of the malarial parasite. Complete recovery rapidly followed hypodermic injections of formiate of quinine.

J. D. Rolleston.

Chyliform pleurisy in a child ('*Bull. et Mém. Soc. Méd. Hôp. de Paris*,' 1913, xxxvi, *p.* 741).—**F. Chevrel** records a case in a boy, aged 5 years, who had been suffering from dyspnœa for several months. He was the subject of adenoids, and probably latent tuberculosis. On paracentesis 550 c.c. of milky fluid were drawn off from the right pleural cavity. Complete recovery took place. The case was remarkable for its insidious onset, slow course and constitutional disturbance. The fluid was alkaline, had a specific gravity of 1020, contained 17·20 per cent. of mineral matter, 20 per cent. of neutral fats, and 55·90 of proteids. Ferments and bacteria were absent. Injection of the fluid into guinea-pigs was negative. No explanation could be given of the pathogeny of the effusion.

J. D. Rolleston.

Purulent pleurisy in childhood (*'La Pediatria,'* 1913, XXI, *p.* 896).—**S. Cannata,** from a study of 88 cases aged from 4 months to 10 years, finds that purulent pleurisy is more frequent in the first twenty-seven years of life, and among males (68 per cent.), and in the great majority of cases is consecutive to lobar pneumonia. In more than half the cases (62 per cent.) Fränkel's diplococcus was found, in a less number (21 per cent.) the streptococcus, while the presence of other pathogenic germs was rare. Objective examination did not yield certain signs of the purulent nature of the exudate, recourse must be had to exploratory puncture in every case. In mild cases, especially if due to Fränkel's diplococcus, absorption of the exudate may be waited for; when it does not tend to disappear or increases, or when the general condition is serious, surgical intervention is called for. Costotomy is preferable since it affords easier evacuation and better drainage. VINCENT DICKINSON.

Pneumococcal empyema in child, aged 7 weeks (*'Arch. de méd. des enf.,'* 1913, XVI, *p.* 850).—**Y. Fouzin.**—The disease came on rapidly with left lobar pneumonia and right broncho-pneumonia. Operation removed a large amount of pus. The case ended fatally.
F. R. B. ATKINSON.

The siphon treatment of empyema in infants and young children compared with other measures—a study of 154 cases (*'Am. Medicine,'* 1913, VIII, *p.* 381).—**E. Holt** finds that (1) empyema in infancy has a high mortality; (2) one of the chief obstacles to recovery is the difficulty in expansion when the lung is subjected to atmospheric pressure; (3) aspiration is not to be depended upon as a means of treatment; (4) the injection of bactericidal substances is open to danger; (5) rib-resection is not advisable in recent acute cases under the age of two years; (6) simple incision between the ribs with the introduction of a single tube is sufficient in most cases to secure adequate drainage; (7) siphon drainage is the best method of drainage. F. R. B. ATKINSON.

Acute non-specific pneumonia in the first days of life (*'Jahrb. f. Kinderheilk.,'* 1914, LXXIX, *p.* 140).—**T. E. H. Thaysen** distinguishes the following forms: (1) Placental infectious pneumonia, due to syphilis, tubercle, pneumococci, streptococci and staphylococci. (2) Aspiration pneumonia resulting from aspiration of fœtid urine, purulent secretion from a pathological condition of the vagina, from secretion from a normal vagina, or from milk or other liquid. (3) Ærogenous. (4) Metastatic resulting from a primary affection of the bowel or navel infection or other lesions. This is excessively rare. The commonest form is the aspiration pneumonia. The aerogenous is rare. The author gives various statistics of cases met with by him of the various forms. F. R. B. ATKINSON.

Acute lobar pneumonia secondary to measles (*'Thèses de Lyon,'* 1912–13, No. 58).—**L. Grand.**—The thesis contains the histories of fourteen cases, twelve of which occurred in children aged from one to ten years, and two in adults. Grand concludes as follows: (1) Acute lobar pneumonia in measles, though less frequent than broncho-pneumonia, should not be regarded as altogether exceptional. (2) Its symptoms and course resemble those of primary pneumonia, but as a rule the signs are less definite and the course is more irregular and protracted. It has also a tendency to grey

hepatisation. (3) Pseudo-lobar broncho-pneumonia often has the same symptoms as those of acute lobar pneumonia. A clinical diagnosis can only be made by radioscopy, when in the case of acute lobar pneumonia an axillary triangle will be found (*vide* British Journal of Children's Diseases, 1913, x, *p.* 318). (4) The pathological diagnosis is made by finding on section a triangular zone of hepatisation and by microscopical examination of the hepatised area. J. D. Rolleston.

The curative value of turpentine subcutaneous injections (*'Lyon Méd.,'* 1913, cxxi, *p.* 941).—**Péhu** and **Pillon** give the results of their investigations on this method of treating broncho-pneumonia. They employed a dose of $\frac{1}{2}$ c.c. in children below 2 years, and 1 c.c. above that age. Pus formation, either manifest or latent, circumscribed or diffuse, occurred without exception. In 78 cases they had 30 cures. In 16 out of 34 cases of broncho-pneumonia the injection was followed by rapid improvement: 13 died. In broncho-pneumonia following diphtheria 13 out of 18 cases were fatal. In that following measles there were 8 deaths and 2 cures. Three cases of broncho-pneumonia following whooping-cough were all cured. Two cases of scarlet fever were not benefited. The authors considered their results as the reverse of encouraging. Vincent Dickinson.

Surgery.

The evolution of surgical tuberculosis in the infant (*'Arch. de méd. des enf.,'* 1914, xvii, *p.* 161).—**Froelich** considers surgical tuberculosis from the time of birth up to the second year and finds that its frequency is very great. Cutaneous gummata may occur as early as the fifteenth day. It usually takes on the hypertrophic form. Fungosities suppurate rapidly, but fistulæ are of short duration. In later childhood it is usually epiphyseal; before that time frequently diaphyseal, attacking both the smaller and larger bones. In the hip, early luxation often results. In the knee, an acute form may develop and resemble an acute infection, and is often quickly cured without stiffness. The disease of the bones is very often multiple, but this does not make the prognosis necessarily more grave. The mortality is about 10 per cent. Tubercular adenitis is of acute origin and leaves few scars. Visceral tuberculosis is less favourable. Peritonitis is very fatal. Tuberculosis of the testicle is very common; the evolution is often acute. Treatment in surgical tuberculosis should be conservative as far as possible.
F. R. B. Atkinson.

Significance of von Pirquet's test in surgical tuberculosis in children (*'Boston. Med. and Surg. Journ.,'* 1914, clxx, *p.* 550).—**L. B. Robertson** records the results of the test made on 350 cases of surgical tuberculosis, all under twelve years of age. Only 2·9 per cent. were negative. Of cases without definite clinical evidence of tuberculosis, although some showed enlarged anterior cervical glands and others gave a family history of tuberculosis, 14 per cent. gave a positive reaction. In apparently non-tuberculous cases the reaction was more frequently negative in the younger children. Thus, of 48 such cases, aged from 0–4 years, 10 per cent. were positive, of 61 cases, aged from 4–8 years, 14·5 per cent. were positive, and of 40 cases, aged from 8–12 years, 17·5 per cent. were positive. J. D. Rolleston.

Multiple bone tuberculosis, atypical in its distribution and X-ray appearances (*'Boston Med. and Surg. Journ.,'* 1914, CLXX, *p.* 547).—**H. J. Fitz Simmons.**—A boy, aged 16 months, presented three tumour masses on the right parietal region, a fluctuating reddened area about the size of a pea at the outer canthus of each eye, a nodular thickening along the descending ramus and angle of the left lower jaw, a few small areas on the right thigh covered with dark crusts, an area 2 in. long and 1 in. wide over the upper third of the right tibia, and swelling of the epiphysis of the radial joint at the elbow. Microscopical examination of a section of the superficial lesions showed typical tubercles. F. R. B. ATKINSON.

Focal tuberculosis with multiple sequestra (*'Arch de Méd. des enf.,'* 1913, XVI, *p.* 921).—**A. Trèves** describes the following lesions in a child three years of age: (1) Ulceration of the first phalanx of the left great toe; (2) swelling of the left first metatarsal bone; (3) swelling and fluctuation of the first phalanx of the right fourth toe; (4) swelling of the left elbow; (5) right elbow much enlarged; (6) first phalanx of the middle left finger increased in size; (7) third metacarpal slightly enlarged; (8) first and second phalanges of the right index enlarged and ulcerated; (9) first phalanx of the right middle finger and the metacarpo-phalangeal articulation much enlarged. Radioscopy showed numerous sequestra in these bones. Operation was performed successfully on the various lesions.

F. R. B. ATKINSON.

Cure of external tuberculosis by means of heliotherapy (*'Paris Méd.,'* 1912–13, II, *p.* 257).—**Rollier** describes his method of treatment of enlarged glands, tuberculosis of joints and open cutaneous tuberculosis by means of fresh air and the sun's rays with numerous drawings of the various lesions thus treated with success, and finds that closed tuberculosis is, as a rule, cured by this method, and frequently also open tuberculosis.

F. R. B. ATKINSON.

The use of celluloid in the treatment of tuberculous disease of the spine (*'Brit. Med. Journ.,'* 1913, I, *p.* 1200).—**H. J. Gauvain** fully describes the technique of making spinal jackets from celluloid. Jackets made of this material are light, elegant, rigid, but slightly elastic, strong, are not acted upon by the heat and the moisture of the body, maintain their shape indefinitely, and are comparatively easy to make. Their greatest value lies in the fact that they may be made to accurately fit a cast of the patient and maintain the shape of this cast indefinitely. The article is illustrated.

J. ALLAN.

Tuberculosis of the genital organs in children (*'Journ. Amer. Med. Assoc.,'* 1913, LXI, *p.* 2051).—**O. Lyons** states that of ninety-three cases of tuberculosis involving the testes in children reported by a number of authors, forty-four were under 2 and forty-seven over 12 years of age, *i. e.* about half the cases occur in the first period of childhood. Generally, the disease is localised in one testicle; however, it often affects both. In the first period of childhood it occurs nearly as often on one side as on the other, but after the age of twelve it occurs more frequently on the left side, as is the case with varicocele. This is thought to be due to disturbed circulation during walking, giving rise to some difference in the return of the venous blood. Tuberculosis of the undescended testicle in infants is very rare. In

some cases the author believes that the male generative organs are the seat of primary tuberculosis, and although such a condition of the prostate and seminal vesicles in children is rare, he reports the case of an infant, aged 20 months, whose mother was tuberculous. The child had lost weight and had frequent micturition and irritability. The urine contained pus, blood and tubercle bacilli and a large, hard mass was found in the region of the prostate and vesicles on the left side. There was no enlargement of the lymphatic glands, but the liver and spleen were increased in size. The child died three and a half months later. At the post-mortem the thoracic cavity was normal, the mesenteric glands were enlarged, soft and caseous, and the liver and spleen were enlarged and contained many small nodules on the surface; the kidneys were normal. In the region of the vesicles was a large mass on the left side, which contained the vesicle and ampulla; it was filled with caseous nodules and abscess cavities. The bladder was normal. Microscopical examination of the mass, liver and spleen proved them to be tuberculous. Primary tuberculosis of the genital organs in children frequently gives rise to no symptoms. Thus a child, aged 7 months, was first noticed to have a swelling in the left side of the scrotum three or four weeks prior to being seen; this had caused no pain or other symptoms. The epididymis was found to be nodular and enlarged to the size of a walnut. Operation was at first refused, but later softening occurred and thick caseous material was evacuated, which inoculation into a guinea-pig proved to be tuberculous. The cavity was packed with gauze and the mass gradually diminished in size, though a fistula remained for some months. The patient made a good recovery, and was in perfect health four and a half years later, the only evidence of his previous trouble being a thick fibrous cord running from the site of the incision to the globus minor. Another case was that of a baby, aged 16 months, who, two or three months previously, was noticed to have a swelling and hardness of the scrotum. The mother was suspected of tuberculosis; there was a history of the disease in her family. The child was found to have a hydrocele with a mass below and behind the sac. The hydrocele was tapped, but the contained straw-coloured fluid was not examined. The epididymis was then found to be involved. Operation was refused, and later, softening of and redness over the mass appeared. An incision was made and pus evacuated, but no tubercle bacilli were found. The cavity was packed and shortly after another abscess was treated in the same way. Four years later the child was in good health, a small, firm band running from the scar to a small nodule in the epididymis, being all that remained. The prognosis of primary tuberculosis of the genitalia in children is usually favourable, and operation should always be advised.

T. R. Whipham.

Tuberculosis of the testicle (*'Med. Record,'* 1914, lxxxv, *p.* 146).—**A. L. Goodman** records a case in a child 2 years and 10 months of age ending in cure. The disease is four times less frequent in children than in adults. The proportion of testicular tuberculosis is one in two hundred cases of infantile tuberculosis. It may be either primary or secondary, and the prognosis is good in the primary form as the disease is restricted, unilateral, and not extending above the inguinal ring. The secondary form is usually more extensive and the remote results very unfavourable. There are numerous references to the literature. F. R. B. Atkinson.

Reviews.

Diagnosen af Barnetuberkulosens kliniske Initialformer (The Diagnosis of the Earliest Clinical Forms of Tuberculosis in Children). By Herman G. Gade. Kristiania: Steen'ske Bogtrykkeri, 1914.

In this monograph, which forms a supplement to the seventy-fifth volume of the 'Norsk Mag. for Laegevidenskaben,' the writer states the well-known fact that in infants and children tuberculosis of the lungs habitually begins as tuberculosis of the lymphatic glands at the roots of the lungs. This condition was recognised clinically as long ago as 1780 by Lalouette; it was very carefully studied by Rilliet and Barthez in 1840, and since that date has often been the subject of clinical and pathological investigation. In 1824 it was distinguished as "bronchial phthisis," and contrasted with "pulmonary phthisis," by Leblond. Before the discovery of the Röntgen rays it was diagnosed by percussion and auscultation. Many of the authorities have believed that pathological enlargement of the glands at the roots of the lungs could be diagnosed with certainty by percussion of the interscapular region of the back; von Korányi and others particularly recommend percussion over the upper dorsal vertebral spines. But other authorities (Widerhofer, Henoch, Heubner, Schlossmann, Sukiennikow) have failed to obtain trustworthy evidence here by percussion. As for auscultation, there is a more general agreement that tuberculosis of the bronchial glands produces changes in the breath-sounds, and also in the voice-sounds according to D'Espine, heard in the interscapular area. Gade's own experience has been gained at the Hagevik Seaside Hospital, where he has seen over 1000 children with various tuberculous infections during the last fifteen years. He attaches little diagnostic importance to the enlarged superficial venules so often seen on the upper part of the thorax in children; they may be evidence of venous stasis, but are not evidence of venous obstruction at or near the roots of the lungs. He is not familiar with the "nervous" cough so often attributed to enlargement of the bronchial glands, and said to be "spastic," "whooping," "*coqueluchöide*" (by the French), "high-pitched," and due to pressure on the vagus nerve. He has not met with attacks of dyspnœa due to these glands, though many German authors have described such. In one case he saw aphonia and tachycardia caused by general and enormous enlargement of the lymphatic glands, due to tuberculosis; the patient, a girl aged 18 years, died in a few days, suffocated by rupture of a large gland into a main bronchus. He has never found Petruschky and Neisser's sign of spinalgia, tenderness on pressure over the second to the seventh dorsal vertebral spines, and places no trust in pains in the chest or under the sternum for the diagnosis of enlargement of the bronchial glands. In the examination of his patients, he looks for interscapular dulness with the patient sitting, his arms loosely crossed in front of him, his elbows resting on his thighs. Gade often finds this dulness, but remarks that while some authorities attribute it to enlargement of the bronchial glands, others say it is due to venous or lymphatic congestion, and yet others describe it as present in the normal person. The same is true of the parasternal dulness, or dulness on either side of the manubrium sterni, so often seen in tuberculous children and attributed by some to enlargement of the tracheobronchial gland. Gade attributes no decisive importance to these areas of

dulness; and he holds that the auscultatory phenomena (prolonged expiration, harsh breath-sounds) heard between the scapulæ are not even approximately certain evidence of enlargement of the bronchial glands. He doubts whether "puerile breath-sounds" are ever heard in normal children free from active or obsolete tuberculosis at the root of the lung. His general conclusion is that neither percussion nor auscultation can determine the presence of enlarged bronchial glands.

The most interesting part of his paper is concerned with the *x*-ray diagnosis of these glands, illustrated by thirteen plates and sixteen silver prints. He notes the very wide variations in the shadows cast by the roots of the lungs in normal persons, the streaks and strands and "birch-broom-like" wisps radiating out from the pulmonary hilus. These streaks are commonly said to be mainly pulmonary blood-vessels in young people, mainly bronchial structures in older people, though it is hard to see why they should vary as widely as they do in different persons. Gade supposes that they may really be strands of connective-tissue for the most part, peribronchial, perivascular, and periglandular, and therefore most marked in the old whose lungs are much more fibrous than those of the young. He believes that it is this fibrous tissue that causes the mottling characteristic of the normal adult lung. He agrees with other authors that mere enlargement (up to, say, four times the normal size) does not suffice to make the bronchial glands visible by use of the *x*-rays, or distinguishable from the vague shadows cast by pulmonary or pleural infiltrations. Until they are calcified, the glands cannot be identified as glands; he gives a skiagram and details of a post-mortem examination illustrating this point. He emphasises the importance of defining the "normal" in skiagrams of the lungs, and of excluding from "the normal" all cases with even obsolete pulmonary (or glandular) tuberculosis. With this object he has tested a number of children with von Pirquet's test and with test injections of tuberculin, and has found thirteen who gave no reaction to either. He concludes that these children were free from tuberculous infection, and gives thirteen admirable silver prints of their thoracograms to serve as standards for the normal. None of these children had any puerile respiration-sounds; seven were healthy children from a Home, six were "tuberculous" patients in his hospital. The first seven skiagrams show indefinite streaks and wisps in plenty, but no so-called "gland-shadows," and no detailed or sharply-defined shadows. The remaining six skiagrams, from "tuberculous" patients clinically free from tuberculosis so far as the point could be determined by tuberculin, showed larger and more opaque shadows at the hilus, with sharper edges, and a few small, well-defined blotches. He classifies the skiagrams of children with pulmonary or bronchial gland tuberculosis into four classes, according as the opacities formed in the lungs are mainly due to: (1) Tuberculous or fibrous tissue in connection with the bronchial or hilar glands; (2) peribronchial and perivascular infiltration, with well-defined streaks; (3) advanced stages of (2), including, *e. g.* tuberculous pneumonia; (4) diffuse tuberculous processes, including miliary tuberculosis. He notes that in practice these various types often run into one another, and illustrates them with skiagraphic plates. His general conclusion is that the *x*-rays fail, as percussion and auscultation fail, to give us a certain method of diagnosing tuberculosis of the bronchial glands; this is particularly the case in the initial stages of the disease, the time at which definite information is most urgently required. But the skiagram can give definite information as to the presence of tuberculosis at the roots of the lungs, even in its earliest

stages, although it cannot absolutely define what tissue it is that is infected. Over one hundred references to the literature are given at the end of this well-written and well-illustrated article. A. J. J.-B.

Acute General Miliary Tuberculosis. By Prof. G. Cornet. Translated by F. S. Tinker, M.B., B.C., etc. London: John Bale, Sons, & Danielsson, Ltd., 1914. Pp. 107. Price 6s. net.

This monograph describes at length the ætiology and pathological anatomy of this disease, and also gives a full consideration to the subject of differential diagnosis, which much enhances the value of the work, as the diagnosis in many instances is by no means easy, and this is perhaps the most valuable chapter in the book. The author finds no treatment of any avail in most instances, but does not look on the disease as entirely hopeless. He mentions one case cured by potassium iodide, and one by Marmorek's serum. We can recommend the work to all interested in the subject. It is well translated. F. R. B. A.

The Pocket Formulary for the Treatment of Disease in Children. By Ludwig Freyberger, J.P., M.D.Vienna, M.R.C.P.Lond., M.R.C.S. Eng. Fourth revised and enlarged edition. Adapted to the British Pharmacopœia. With an appendix on Poisons, their Symptoms and Treatment. London: William Heinemann, 1914. Price 7s. 6d. net.

This little work, which is intended for the busy practitioner and senior student, is divided into two main parts and several appendices. The first part contains an alphabetical list of drugs with a description of their properties, use, therapeutics, dose and incompatibles. In numerous instances methods of correcting their taste are given and in the case of poisonous drugs the antidotes are added. The second part is a therapeutic index in which the various diseases are arranged alphabetically with their appropriate drugs and the drugs are classified according to their action. An appendix which is inserted between the first and second parts contains the formulæ for various baths, preparations suitable for gargles, hypodermic injections, inhalations and sprays, nasal, pharyngeal and laryngeal applications, and rectal injections as well as a description of von Pirquet's test and tuberculin treatment. Eighteen pages at the end of the work are devoted to the symptoms and treatment of poisons.

Dr. Freyberger is to be congratuated for his skilful arrangement of so much valuable information within a small compass. The book is well printed and tastefully bound. J. D. R.

Transactions of the American Pediatric Society. Vol. XXV. Edited by L. E. La Fétra, M.D. Chicago: American Medical Association Press.

This volume of transactions of the American Pediatric Society for 1913 contains a large number of interesting papers, many of which are illustrated. Dr. Langley Porter writes on "Pancreatic Insufficiency in Children," a condition which in this country is usually termed "cœliac disease." Dr. L. E. Holt has a paper on "Duodenal Ulcers in Infancy," which includes a summary of the literature on the subject. Dr. T. M. Rotch describes "Three Types of Occlusion of the Œsophagus in Early Life," and Drs. G. R. Pisek and L. T. Le Wald contribute "Further Studies of the Anatomy and Physiology of the Infant Stomach." Other papers which deserve special notice are "Studies on the Incubation Period of Serum Disease," by Dr. D. M. Cowie, and "Acid Intoxication in Children," by Dr. I. A. Abt. T. R. W.